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Set     Items   Description
S1       1535   (HYPERTENSI?) AND (PSGL? OR P(W)SELECTIN OR PADGEM OR GMP(-
            W)140 OR GMP140)
S2        341   (HYPERTENSI?) AND (PSGL? OR P(W)SELECTIN OR PADGEM OR GMP(-
            W)140 OR GMP140)(10N)(INHIBIT? OR ANTAGONI? OR SUPPRESS? OR T-
            REAT? OR THERAP?)
S3        210   RD S2  (unique items)
? s s1 and py<2001
Processing
Processing
Processed  10 of  25 files ...
Processing
Processing
>>>One or more prefixes are unsupported
>>>  or undefined in one or more files.
Processed  20 of  25 files ...
Processing
Completed processing all files
            1535  S1
       106616338  PY<2001
      S4     384  S1 AND PY<2001
? rd s4
      S5     216  RD S4  (unique items)
? s s5 and (atherosclerosis)
             216  S5
          459148  ATHEROSCLEROSIS
      S6      45  S5 AND (ATHEROSCLEROSIS)
? t s6/3/all

 6/3/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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15934018   BIOSIS NO.: 200100105857
Selectins level may be useful clinical markers for endothelial damage in
  hypertension
AUTHOR: Sanada Hironobu (Reprint); Midorikawa Sanae (Reprint); Hashimoto
  Shigeatsu (Reprint); Ohashi Hiroko (Reprint); Watanabe Hidetsuna; Hayashi
  Yoshimitsu; Katoh Tetsuo; Watanabe Tsuyoshi
AUTHOR ADDRESS: Fukushima Medical Univ, Fukushima, Japan**Japan
JOURNAL: Circulation  102 (18 Supplement): pII.321 October 31, 2000
2000
MEDIUM: print
CONFERENCE/MEETING: Abstracts from American Heart Association Scientific
Sessions 2000  New Orleans, Louisiana, USA  November 12-15, 2000; 20001112
SPONSOR: American Heart Association
ISSN: 0009-7322
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English


 6/3/2     (Item 2 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2008 The Thomson Corporation. All rts. reserv.

15933873   BIOSIS NO.: 200100105712
Markers of haemostasis and risk stratification in hypertension using
  the Framingham equation
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AUTHOR: Spencer C (Reprint); Gurney D (Reprint); Blann A (Reprint); Beevers D (Reprint); Lip G (Reprint)
AUTHOR ADDRESS: Haemostasis, Thrombosis and Vascular Biology Unit, University Department of Medecine, City Hospital, Birmingham, UK**UK
JOURNAL: Journal of Hypertension 18 (Suppl. 4): pS149-S150 2000 ***2000***
MEDIUM: print
CONFERENCE/MEETING: 18th Scientific Meeting of the International Society of Hypertension Chicago, Illinois, USA August 20-24, 2000; 20000820
SPONSOR: International Society of Hypertension
ISSN: 0263-6352
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

6/3/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15804991 BIOSIS NO.: 200000523304
Angiotensin II induces leukocyte-endothelial cell interactions in vivo via AT1 and AT2 receptor-mediated P-selectin upregulation
AUTHOR: Piqueras Laura; Kubes Paul; Alvarez Angeles; O'Connor Enrique; Issekutz Andrew C; Esplugues Juan V; Sanz Maria-Jesus (Reprint)
AUTHOR ADDRESS: Departamento de Farmacologia, Facultad de Medicina, Av. Blasco Ibanez, 15-17, 46010, Valencia, Spain**Spain
JOURNAL: Circulation 102 (17): p2118-2123 October 24, 2000 2000
MEDIUM: print
ISSN: 0009-7322
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

6/3/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15603886 BIOSIS NO.: 200000322199
Soluble P-selectin: A marker of platelet activation and vessel wall injury. Increase of soluble ***P*** - ***selectin*** in the blood plasma of patients with myocardial infarction, massive atherosclerosis and primary pulmonary hypertension
AUTHOR: Semenov A V; Kagan-Ponomarev M Ya; Ruda M Ya; Komarov A L; Panchenko E P; Chazova I E; Mazurov A V
JOURNAL: Terapevticheskii Arkhiv 72 (4): p15-20 2000 2000
MEDIUM: print
ISSN: 0040-3660
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: Russian

6/3/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15593685 BIOSIS NO.: 200000311998
Endothelial function and hemostasis
AUTHOR: Becker B F (Reprint); Heindl B; Kupatt C; Zahler S
AUTHOR ADDRESS: Dept. of Physiology, University of Munich, Pettenkofer Str.

12, D-80336, Munich, Germany**Germany
JOURNAL: Zeitschrift fuer Kardiologie 89 (3): p160-167 March, 2000 2000
MEDIUM: print
ISSN: 0300-5860
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

6/3/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15565051 BIOSIS NO.: 200000283364
Vascular endothelial cell activation associated with increased plasma asymmetric dimethyl arginine in children and young adults with hypertension: A basis for atheroma?
AUTHOR: Goonasekera Chulananda D A; Shah Vanita; Rees D D; Dillon Michael J (Reprint)
AUTHOR ADDRESS: Nephrourology Unit, Institute of Child Health, 30 Guilford Street, London, WC1 N1EH, UK**UK
JOURNAL: Blood Pressure 9 (1): p16-21 2000 2000
MEDIUM: print
ISSN: 0803-7051
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

6/3/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15341208 BIOSIS NO.: 200000059521
Endothelin-1 causes P-selectin-dependent leukocyte rolling and adhesion within rat mesenteric microvessels
AUTHOR: Sanz Maria-Jesus (Reprint); Johnston Brent; Issekutz Andrew; Kubes Paul
AUTHOR ADDRESS: Departamento de Farmacologia, Facultat de Medicina, Universitat de Valencia, Av. Blasco Ibanez, 15-17, 46010, Valencia, Spain **Spain
JOURNAL: American Journal of Physiology 277 (5 PART 2): pH1823-H1830 Nov., 1999 1999
MEDIUM: print
ISSN: 0002-9513
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

6/3/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14918499 BIOSIS NO.: 199900178159
Levels of soluble cell adhesion molecules in patients with angiographically defined coronary atherosclerosis
AUTHOR: Saku Keijiro (Reprint); Zhang Bo; Ohta Takao; Shirai Kazuyuki; Tsuchiya Yoshihiro; Arakawa Kikuo
AUTHOR ADDRESS: Department of Internal Medicine, Fukuoka University School

of Medicine, 7-45-1 Nanakuma Jonan-ku, Fukuoka, 814-0180, Japan**Japan
JOURNAL: Japanese Circulation Journal 63 (1): p19-24 Jan., 1999 ***1999***
MEDIUM: print
ISSN: 0047-1828
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

6/3/9 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14278298 BIOSIS NO.: 199800072545
Relation of endothelium, thrombogenesis, and hemorheology in systemic hypertension to ethnicity and left ventricular hypertrophy
AUTHOR: Lip Gregory Y H (Reprint); Blann Andrew D; Jones Alan F; Lip Peck Lin; Beevers D Gareth
AUTHOR ADDRESS: Haemostasis Thrombosis and Vasc. Biol. Unit, Univ. Dep. Med., City Hosp., Birmingham B18 7QH, UK**UK
JOURNAL: American Journal of Cardiology 80 (12): p1566-1571 Dec. 15, 1997 1997
MEDIUM: print
ISSN: 0002-9149
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

6/3/10 (Item 10 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13834186 BIOSIS NO.: 199799468246
Circulating vascular cell adhesion molecule-1 correlates with the extent of human atherosclerosis in contrast to circulating intercellular adhesion molecule-1, E-selectin, P-selectin, and thrombomodulin
AUTHOR: Peter Karlheinz (Reprint); Nawroth Peter; Conradt Christian; Nordt Thomas; Weiss Thomas; Boehme Michael; Wunsch Andreas; Allenberg Jens; Kuebler Wolfgang; Bode Christoph
AUTHOR ADDRESS: Internal Med. III, Univ. Heidelberg, Bergheimer Strasse 58, 69115 Heidelberg, Germany**Germany
JOURNAL: Arteriosclerosis Thrombosis and Vascular Biology 17 (3): p505-512 1997 1997
ISSN: 1079-5642
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

6/3/11 (Item 11 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13207437 BIOSIS NO.: 199698675270
Soluble adhesion molecule P-selectin and endothelial dysfunction in essential hypertension: Implications for atherogenesis? A preliminary report
AUTHOR: Lip Gregory Y H (Reprint); Blann Andrew D; Zarifis John; Beevers Michele; Lip Peck-Lin; Beevers D Gareth

AUTHOR ADDRESS: Univ. Dep. Medicine, City Hosp., Dudley Rd., Birmingham B18 7QH, UK**UK
JOURNAL: Journal of Hypertension 13 (12 PART 2): p1674-1678 1995 1995
ISSN: 0263-6352
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

6/3/12 (Item 12 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12934590 BIOSIS NO.: 199598402423
Platelet-derived microparticles may influence the development of atherosclerosis in diabetes mellitus
AUTHOR: Nomura Shosaku (Reprint); Suzuki Masahiko; Katsura Kaoruko; Xie Gui Lan; Miyazaki Yasuhiko; Miyake Tetsuya; Kido Hirofumi; Kagawa Hideo; Fukuhara Shirou
AUTHOR ADDRESS: The First Dep. Internal Med., Kansai Med. Univ., 10-15, Fumizono-cho, Moriguchi, Osaka 570, Japan**Japan
JOURNAL: Atherosclerosis 116 (2): p235-240 1995 1995
ISSN: 0021-9150
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

6/3/13 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

09085737 Genuine Article#: 365XG No. References: 26
Title: Angiotensin II induces leukocyte-endothelial cell interactions in vivo via AT(1) and AT(2) receptor-mediated P-selectin upregulation
Author(s): Piqueras L; Kubes P; Alvarez A; OConnor E; Issekutz AC; Esplugues JV; Sanz MJ (REPRINT)
Corporate Source: UNIV VALENCIA,FAC MED, DEPT FARMACOL, AV BLASCO IBANEZ 15-17/VALENCIA 46010//SPAIN/ (REPRINT); UNIV VALENCIA,FAC MED, DEPT FARMACOL/VALENCIA 46010//SPAIN/; UNIV VALENCIA,DEPT BIOCHEM/VALENCIA 46010//SPAIN/; UNIV CALGARY,IMMUNOL RES GRP/CALGARY/AB T2N 1N4/CANADA/; DALHOUSIE UNIV,DEPT PEDIAT/HALIFAX/NS/CANADA/; DALHOUSIE UNIV,DEPT PATHOL/HALIFAX/NS/CANADA/; DALHOUSIE UNIV,DEPT MICROBIOL/HALIFAX/NS/CANADA/; DALHOUSIE UNIV,DEPT IMMUNOL/HALIFAX/NS/CANADA/
Journal: CIRCULATION, 2000, V102, N17 (OCT 24), P2118-2123
ISSN: 0009-7322 Publication date: 20001024
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621
Language: English Document Type: ARTICLE (ABSTRACT AVAILABLE)

6/3/14 (Item 2 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

08870536 Genuine Article#: 339BB No. References: 121
Title: Testing for endothelial dysfunction
Author(s): Raitakari OT (REPRINT) ; Celermajer DS

Corporate Source: UNIV TURKU,CENT HOSP, TURKU PET CTR, POB 52/FIN-20520 TURKU//FINLAND/ (REPRINT); ROYAL PRINCE ALFRED HOSP,DEPT CARDIOL/SYDNEY/NSW/AUSTRALIA/; UNIV TURKU,DEPT CLIN PHYSIOL/TURKU//FINLAND/; UNIV TURKU,TURKU PET CTR/TURKU//FINLAND/; UNIV SYDNEY,DEPT MED/SYDNEY/NSW 2006/AUSTRALIA/
Journal: ANNALS OF MEDICINE, 2000, V32, N5 (JUL), P293-304
ISSN: 0785-3890 Publication date: 20000700
Publisher: ROYAL SOC MEDICINE PRESS LTD, 1 WIMPOLE STREET, LONDON W1M 8AE, ENGLAND
Language: English Document Type: REVIEW (ABSTRACT AVAILABLE)

6/3/15 (Item 3 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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07920918 Genuine Article#: 224JM No. References: 26
Title: Adhesion molecules in cerebrovascular diseases - Evidence for an inflammatory endothelial activation in cerebral large- and small-vessel disease
Author(s): Fassbender K (REPRINT) ; Bertsch T; Mielke O; Muhlhauser F; Hennerici M
Corporate Source: UNIV HEIDELBERG,KLINIKUM MANNHEIM, DEPT NEUROL, THEODOR KUTZER UFER 1-3/D-68135 MANNHEIM//GERMANY/ (REPRINT); UNIV HEIDELBERG,KLINIKUM MANNHEIM, INST CLIN CHEM/D-68135 MANNHEIM//GERMANY/
Journal: STROKE, 1999, V30, N8 (AUG), P1647-1650
ISSN: 0039-2499 Publication date: 19990800
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 227 EAST WASHINGTON SQ, PHILADELPHIA, PA 19106
Language: English Document Type: ARTICLE (ABSTRACT AVAILABLE)

6/3/16 (Item 4 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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07852096 Genuine Article#: 216BG No. References: 35
Title: Cross-sectional study of soluble intercellular adhesion molecule-1 and cardiovascular risk factors in apparently healthy men
Author(s): Rohde LEP; Hennekens CH; Ridker PM (REPRINT)
Corporate Source: BRIGHAM & WOMENS HOSP,DIV CARDIOVASC, 75 FRANCIS ST/BOSTON//MA/02115 (REPRINT); BRIGHAM & WOMENS HOSP,DIV CARDIOVASC/BOSTON//MA/02115; BRIGHAM & WOMENS HOSP,DIV PREVENT MED/BOSTON//MA/02115; HARVARD UNIV,SCH MED, DEPT AMBULATORY CARE & PREVENT/BOSTON//MA/
Journal: ARTERIOSCLEROSIS THROMBOSIS AND VASCULAR BIOLOGY, 1999, V19, N7 (JUL), P1595-1599
ISSN: 1079-5642 Publication date: 19990700
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 227 EAST WASHINGTON SQ, PHILADELPHIA, PA 19106
Language: English Document Type: ARTICLE (ABSTRACT AVAILABLE)

6/3/17 (Item 5 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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07493240 Genuine Article#: 173CJ No. References: 48
Title: Fosinopril decreases levels of soluble vascular cell adhesion molecule-1 in borderline hypertensive type II diabetic patients with microalbuminuria

Author(s): Gasic S (REPRINT) ; Wagner OF; Fasching P; Ludwig C; Veitl M; Kapiotis S; Jilma B
Corporate Source: UNIV HOSP VIENNA,DIV ENDOCRINOL & METAB, DEPT INTERNAL MED 3, WAHRINGERGURTEL 18-20/A-1090 VIENNA//AUSTRIA/ (REPRINT); UNIV HOSP VIENNA,DEPT CLIN PHARMACOL, ADHES RES GRP ELABORATING THERAPEUT/A-1090 VIENNA//AUSTRIA/; UNIV HOSP VIENNA,MED & CHEM LAB DIAGNOST, INST CLIN/A-1090 VIENNA//AUSTRIA/; UNIV LEIPZIG,DEPT CLIN CHEM & PATHOBIOCHEM/D-7010 LEIPZIG//GERMANY/
Journal: AMERICAN JOURNAL OF HYPERTENSION, 1999, V12, N2,1 (FEB), P 217-222
ISSN: 0895-7061 Publication date: 19990200
Publisher: ELSEVIER SCIENCE INC, 655 AVENUE OF THE AMERICAS, NEW YORK, NY 10010
Language: English Document Type: ARTICLE (ABSTRACT AVAILABLE)

6/3/18 (Item 6 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2008 The Thomson Corp. All rts. reserv.

07309090 Genuine Article#: 145LL No. References: 44
Title: The influence of acute smoking on leucocytes, platelets and the endothelium
Author(s): Blann AD (REPRINT) ; Kirkpatrick U; Devine C; Naser S; McCollum CN
Corporate Source: UNIV BIRMINGHAM,CITY HOSP, DEPT MED, HAEMOSTASIS THROMBOSIS & VASC BIOL UNIT/BIRMINGHAM B18 7QH/W MIDLANDS/ENGLAND/ (REPRINT); UNIV S MANCHESTER HOSP,DEPT SURG/MANCHESTER M20 8LR/LANCS/ENGLAND/
Journal: ATHEROSCLEROSIS, 1998, V141, N1 (NOV), P133-139
ISSN: 0021-9150 Publication date: 19981100
Publisher: ELSEVIER SCI IRELAND LTD, CUSTOMER RELATIONS MANAGER, BAY 15, SHANNON INDUSTRIAL ESTATE CO, CLARE, IRELAND
Language: English Document Type: ARTICLE (ABSTRACT AVAILABLE)

6/3/19 (Item 7 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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07220837 Genuine Article#: 138QD No. References: 36
Title: Circulating cell adhesion molecules are correlated with ultrasound-based assessment of carotid atherosclerosis
Author(s): Rohde LE; Lee RT; Rivero J; Jamacochian M; Arroyo LH; Briggs W; Rifai N; Libby P; Creager MA; Ridker PM (REPRINT)
Corporate Source: BRIGHAM & WOMENS HOSP,DIV CARDIOVASC, DEPT MED, 75 FRANCIS ST/BOSTON//MA/02115 (REPRINT); BRIGHAM & WOMENS HOSP,DIV CARDIOVASC, DEPT MED/BOSTON//MA/02115; HARVARD UNIV,SCH MED, CHILDRENS HOSP, DEPT LAB MED/BOSTON//MA/
Journal: ARTERIOSCLEROSIS THROMBOSIS AND VASCULAR BIOLOGY, 1998, V18 , N11 (NOV), P1765-1770
ISSN: 1079-5642 Publication date: 19981100
Publisher: WILLIAMS & WILKINS, 351 WEST CAMDEN ST, BALTIMORE, MD 21201-2436
Language: English Document Type: ARTICLE (ABSTRACT AVAILABLE)

6/3/20 (Item 8 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2008 The Thomson Corp. All rts. reserv.

06430023 Genuine Article#: YT137 No. References: 39

Title: Putative role of adhesion molecules in metabolic disorders
Author(s): Wagner OF (REPRINT) ; Jilma B
Corporate Source: UNIV LEIPZIG,DEPT CLIN CHEM & PATHOBIOCHEM, PAUL LIST STR 13/D-04103 LEIPZIG//GERMANY/ (REPRINT); UNIV HOSP VIENNA,SCH MED, ADHES RES GRP ELABORATING THERAPEUT, DEPT CLIN PHARMACOL, TARGET/VIENNA//AUSTRIA/
Journal: HORMONE AND METABOLIC RESEARCH, 1997, V29, N12 (DEC), P 627-630
ISSN: 0018-5043 Publication date: 19971200
Publisher: GEORG THIEME VERLAG, P O BOX 30 11 20, D-70451 STUTTGART, GERMANY
Language: English Document Type: ARTICLE (ABSTRACT AVAILABLE)

6/3/21 (Item 9 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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06349615 Genuine Article#: YL375 No. References: 61
Title: Circulating adhesion molecules VCAM-1, ICAM-1, and E-selectin in carotid atherosclerosis and incident coronary heart disease cases - The atherosclerosis risk in communities (ARIC) study
Author(s): Hwang SJ; Ballantyne CM; Sharrett AR; Smith LC; Davis CE; Gotto AM; Boerwinkle E (REPRINT)
Corporate Source: UNIV TEXAS,HLTH SCI CTR, CTR HUMAN GENET, POB 20334/HOUSTON//TX/77225 (REPRINT); UNIV TEXAS,HLTH SCI CTR, CTR HUMAN GENET/HOUSTON//TX/77225; UNIV TEXAS,HLTH SCI CTR, INST MOL MED/HOUSTON//TX/77225; BAYLOR COLL MED,DEPT MED/HOUSTON//TX/77030; NHLBI,EPIDEMIOL & BIOMETRY PROGRAM/BETHESDA//MD/20892; UNIV N CAROLINA,SCH PUBL HLTH, DEPT BIOSTAT/CHAPEL HILL//NC/
Journal: CIRCULATION, 1997, V96, N12 (DEC 16), P4219-4225
ISSN: 0009-7322 Publication date: 19971216
Publisher: AMER HEART ASSOC, 7272 GREENVILLE AVENUE, DALLAS, TX 75231-4596
Language: English Document Type: ARTICLE (ABSTRACT AVAILABLE)

6/3/22 (Item 10 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2008 The Thomson Corp. All rts. reserv.

06221361 Genuine Article#: YC877 No. References: 69
Title: Endothelial cell injury in cardiovascular surgery: An overview
Author(s): Verrier ED (REPRINT) ; Boyle EM
Corporate Source: UNIV WASHINGTON,DEPT SURG, DIV CARDIOTHORAC SURG, 1059 PACIFIC AVE NE, BOX 356310/SEATTLE//WA/98195 (REPRINT)
Journal: ANNALS OF THORACIC SURGERY, 1997, V64, N4,S (OCT), PS2-S8
ISSN: 0003-4975 Publication date: 19971000
Publisher: ELSEVIER SCIENCE INC, 655 AVENUE OF THE AMERICAS, NEW YORK, NY 10010
Language: English Document Type: ARTICLE (ABSTRACT AVAILABLE)

6/3/23 (Item 11 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2008 The Thomson Corp. All rts. reserv.

05962307 Genuine Article#: XK575 No. References: 37
Title: Anticardiolipin antibodies are not associated with restenosis or endothelial activation after percutaneous transluminal angioplasty
Author(s): Tsakiris DA (REPRINT) ; Tschopl M; Jager K; Wolf F; Marbet GA
Corporate Source: UNIV BASEL HOSP,HAEMOSTASIS LAB, DEPT CENT LAB/CH-4031

BASEL//SWITZERLAND/ (REPRINT)
Journal: INTERNATIONAL ANGIOLOGY, 1997, V16, N2 (JUN), P88-93
ISSN: 0392-9590 Publication date: 19970600
Publisher: EDIZIONI MINERVA MEDICA, CORSO BRAMANTE 83-85 INT JOURNALS DEPT., 10126 TURIN, ITALY
Language: English Document Type: ARTICLE (ABSTRACT AVAILABLE)

6/3/24 (Item 12 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2008 The Thomson Corp. All rts. reserv.

05920616 Genuine Article#: XG541 No. References: 104
Title: Vascular homeostasis, adhesion molecules, and macrovascular disease in non-insulin-dependent diabetes mellitus
Author(s): Carter AM; Grant PJ (REPRINT)
Corporate Source: UNIV LEEDS,RES SCH MED, UNIT MOL VASC MED, LEEDS GEN INFIRM, G FLOOR, MARTIN WING/LEEDS LS1 3EX/W YORKSHIRE/ENGLAND/ (REPRINT); UNIV LEEDS,RES SCH MED, UNIT MOL VASC MED, LEEDS GEN INFIRM/LEEDS LS1 3EX/W YORKSHIRE/ENGLAND/
Journal: DIABETIC MEDICINE, 1997, V14, N6 (JUN), P423-432
ISSN: 0742-3071 Publication date: 19970600
Publisher: JOHN WILEY & SONS LTD, BAFFINS LANE CHICHESTER, W SUSSEX, ENGLAND PO19 1UD
Language: English Document Type: REVIEW (ABSTRACT AVAILABLE)

6/3/25 (Item 13 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2008 The Thomson Corp. All rts. reserv.

05847709 Genuine Article#: XB411 No. References: 38
Title: Leukocyte activation in atherosclerosis: Correlation with risk factors
Author(s): Elneihoum AM (REPRINT) ; Falke P; Hedblad B; Lindgarde F; Ohlsson K
Corporate Source: UNIV LUND HOSP,DEPT MED/S-20502 MALMO//SWEDEN/ (REPRINT); UNIV LUND HOSP,DEPT SURG PATHOPHYSIOL/S-20502 MALMO//SWEDEN/
Journal: ATHEROSCLEROSIS, 1997, V131, N1 (MAY), P79-84
ISSN: 0021-9150 Publication date: 19970500
Publisher: ELSEVIER SCI IRELAND LTD, CUSTOMER RELATIONS MANAGER, BAY 15, SHANNON INDUSTRIAL ESTATE CO, CLARE, IRELAND
Language: English Document Type: ARTICLE (ABSTRACT AVAILABLE)

6/3/26 (Item 14 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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05784955 Genuine Article#: WX244 No. References: 119
Title: Review article: Platelets in inflammatory bowel disease - Pathogenetic role and therapeutic implications
Author(s): Collins CE (REPRINT) ; Rampton DS
Corporate Source: ST MARYS HOSP,DEPT MED/LONDON W2//ENGLAND/ (REPRINT); ST BARTHOLOMEWS & ROYAL LONDON SCH MED & DENT,GI SCI RES UNIT/LONDON//ENGLAND/
Journal: ALIMENTARY PHARMACOLOGY & THERAPEUTICS, 1997, V11, N2 (APR), P237-247
ISSN: 0269-2813 Publication date: 19970400
Publisher: BLACKWELL SCIENCE LTD, OSNEY MEAD, OXFORD, OXON, ENGLAND OX2 0EL
Language: English Document Type: REVIEW (ABSTRACT AVAILABLE)

6/3/27 (Item 15 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2008 The Thomson Corp. All rts. reserv.

05624757 Genuine Article#: WL697 No. References: 187
Title: Vascular endothelial dysfunction
Author(s): DeMeyer GRY (REPRINT) ; Herman AG
Corporate Source: UNIV INSTELLING ANTWERP,DIV PHARMACOL, UNIV PL 1/B-2610 WILRIJK//BELGIUM/ (REPRINT)
Journal: PROGRESS IN CARDIOVASCULAR DISEASES, 1997, V39, N4 (JAN-FEB) , P325-342
ISSN: 0033-0620 Publication date: 19970100
Publisher: W B SAUNDERS CO, INDEPENDENCE SQUARE WEST CURTIS CENTER, STE 300, PHILADELPHIA, PA 19106-3399
Language: English Document Type: REVIEW

6/3/28 (Item 16 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2008 The Thomson Corp. All rts. reserv.

05574338 Genuine Article#: WH486 No. References: 115
Title: Nitric oxide synthase: Role in the genesis of vascular disease
Author(s): Cooke JP (REPRINT) ; Dzau VJ
Corporate Source: STANFORD UNIV,DIV CARDIOVASC MED/STANFORD//CA/94305 (REPRINT)
Journal: ANNUAL REVIEW OF MEDICINE, 1997, V48, P489-509
ISSN: 0066-4219 Publication date: 19970000
Publisher: ANNUAL REVIEWS INC, 4139 EL CAMINO WAY, PO BOX 10139, PALO ALTO, CA 94303-0139
Language: English Document Type: REVIEW (ABSTRACT AVAILABLE)

6/3/29 (Item 17 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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05484950 Genuine Article#: WB840 No. References: 37
Title: OXIDIZED LOW-DENSITY LIPOPROTEINS AND MICROVASCULAR RESPONSES TO ISCHEMIA-REPERFUSION
Author(s): LIAO LX; HARRIS NR; GRANGER DN
Corporate Source: LOUISIANA STATE UNIV,MED CTR,DEPT PHYSIOL & BIOPHYS,1501 KINGS HIGHWAY,POB 33932/SHREVEPORT//LA/71130; LOUISIANA STATE UNIV,MED CTR,DEPT PHYSIOL & BIOPHYS/SHREVEPORT//LA/71130
Journal: AMERICAN JOURNAL OF PHYSIOLOGY-HEART AND CIRCULATORY PHYSIOLOGY, 1996, V40, N6 (DEC), PH2508-H2514
ISSN: 0363-6135
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

6/3/30 (Item 18 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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05358261 Genuine Article#: BG58X No. References: 621
Title: DIFFERENTIATED PROPERTIES AND PROLIFERATION OF ARTERIAL SMOOTH-MUSCLE CELLS IN CULTURE
Author(s): THYBERG J
Corporate Source: KAROLINSKA INST,DEPT MOL & CELL BIOL/S-17177

STOCKHOLM//SWEDEN/
Journal: INTERNATIONAL REVIEW OF CYTOLOGY-A SURVEY OF CELL BIOLOGY, 1996, V169, P183-265
ISSN: 0074-7696
Language: ENGLISH Document Type: REVIEW

6/3/31 (Item 19 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2008 The Thomson Corp. All rts. reserv.

05230098 Genuine Article#: VJ676 No. References: 54
Title: EXACERBATION OF ATHEROSCLEROSIS BY HYPERTENSION - POTENTIAL MECHANISMS AND CLINICAL IMPLICATIONS
Author(s): CHOBANIAN AV; ALEXANDER RW
Corporate Source: BOSTON UNIV,SCH MED,80 E CONCORD ST/BOSTON//MA/02118; EMORY UNIV,SCH MED/ATLANTA//GA/00000
Journal: ARCHIVES OF INTERNAL MEDICINE, 1996, V156, N17 (SEP 23), P 1952-1956
ISSN: 0003-9926
Language: ENGLISH Document Type: REVIEW (Abstract Available)

6/3/32 (Item 20 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2008 The Thomson Corp. All rts. reserv.

05172790 Genuine Article#: VE769 No. References: 44
Title: INCREASE IN NUMBER OF WEIBEL-PALADE BODIES AND ENDOTHELIN-1 RELEASE FROM ENDOTHELIAL-CELLS IN THE CADMIUM-TREATED RAT THORACIC AORTA
Author(s): DOI Y; OZAKA T; FUKUSHIGE H; FURUKAWA H; YOSHIZUKA M; FUJIMOTO S
Corporate Source: UNIV OCCUPAT & ENVIRONM HLTH,SCH MED,DEPT ANAT/KITAKYUSHU/FUKUOKA 807/JAPAN/; UNIV OCCUPAT & ENVIRONM HLTH,SCH HLTH SCI,DEPT NURSING/KITAKYUSHU/FUKUOKA 807/JAPAN/; KURUME UNIV,SCH MED,DEPT ANAT/KURUME/FUKUOKA 830/JAPAN/
Journal: VIRCHOWS ARCHIV-AN INTERNATIONAL JOURNAL OF PATHOLOGY, 1996 , V428, N6 (AUG), P367-373
ISSN: 0945-6317
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

6/3/33 (Item 21 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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05170303 Genuine Article#: VE928 No. References: 59
Title: HYPERTENSION, THE ENDOTHELIUM AND THE PATHOGENESIS OF CHRONIC VASCULAR-DISEASE
Author(s): HALLER H
Corporate Source: HUMBOLDT UNIV BERLIN,VIRCHOW KLINIKUM,FRANZ VOLHARD KLIN,WILTBERG STR 50/D-13125 BERLIN//GERMANY/
Journal: KIDNEY & BLOOD PRESSURE RESEARCH, 1996, V19, N3-4, P166-171
ISSN: 1420-4096
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

6/3/34 (Item 22 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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05042811 Genuine Article#: TL380 No. References: 44

Title: DNA POLYMORPHISMS IN ADHESION MOLECULE GENES - A NEW RISK FACTOR FOR EARLY ATHEROSCLEROSIS
Author(s): WENZEL K; ERNST M; ROHDE K; BAUMANN G; SPEER A
Corporate Source: CHARITE,DEPT INTERNAL MED 1,DIV MOLEC BIOL,ZIEGELSTR 5-9/D-10117 BERLIN//GERMANY/; HUMBOLDT UNIV BERLIN,CHARITE,DEPT INTERNAL MED 1,DIV MOLEC BIOL/D-10098 BERLIN//GERMANY/; MAX DELBRUCK CTR MOLEC MED/D-13122 BERLIN//GERMANY/
Journal: HUMAN GENETICS, 1996, V97, N1 (JAN), P15-20
ISSN: 0340-6717
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

6/3/35 (Item 23 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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04960932 Genuine Article#: UV164 No. References: 34
Title: EVIDENCE FOR ACTIVATION OF ENDOTHELIUM AND MONOCYTES IN HYPERTENSIVE RATS
Author(s): LIU Y; LIU TN; MCCARRON RM; SPATZ M; FEUERSTEIN G; HALLENBECK JM ; SIREN AL
Corporate Source: UNIFORMED SERV UNIV HLTH SCI,DEPT NEUROL,4301 JONES BRIDGE RD/BETHESDA//MD/20814; UNIFORMED SERV UNIV HLTH SCI,DEPT NEUROL/BETHESDA//MD/20814
Journal: AMERICAN JOURNAL OF PHYSIOLOGY-HEART AND CIRCULATORY PHYSIOLOGY, 1996, V39, N6 (JUN), PH2125-H2131
ISSN: 0363-6135
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

6/3/36 (Item 24 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2008 The Thomson Corp. All rts. reserv.

04805720 Genuine Article#: UJ126 No. References: 25
Title: HYPERTENSION-ENHANCED MONOCYTE ADHESION IN EXPERIMENTAL ATHEROSCLEROSIS
Author(s): TROPEA BI; HUIE P; COOKE JP; TSAO PS; SIBLEY RK; ZARINS CK
Corporate Source: STANFORD UNIV HOSP,DIV VASC SURG,SUITE H-3600/STANFORD//CA/94305; STANFORD UNIV,SCH MED,DIV VASC SURG/STANFORD//CA/94305; STANFORD UNIV,SCH MED,DEPT PATHOL/STANFORD//CA/94305; STANFORD UNIV,SCH MED,DIV CARDIOVASC MED/STANFORD//CA/94305
Journal: JOURNAL OF VASCULAR SURGERY, 1996, V23, N4 (APR), P596-605
ISSN: 0741-5214
Language: ENGLISH Document Type: ARTICLE (Abstract Available)

6/3/37 (Item 1 from file: 45)
DIALOG(R)File 45:EMCare
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00822422 EMCare No: 30786493
Angiotensin II induces leukocyte-endothelial cell interactions in vivo via ATSUB1 and ATSUB2 receptor-mediated P-selectin upregulation
Piqueras L.; Kubes P.; Alvarez A.; O'Connor E.; Issekutz A.C.; Esplugues J.V.; Sanz M.-J.
Dr. M.-J. Sanz, Departamento de Farmacologia, Facultad de Medicina, Av. Blasco Ibanez, 15-17, 46010 Valencia Spain
AUTHOR EMAIL: maria.j.sanz@uv.es
Circulation (CIRCULATION) (United States) 24 OCT 2000, 102/17

(2118-2123)
CODEN: CIRCA ISSN: 0009-7322
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 26
RECORD TYPE: Abstract

6/3/38 (Item 1 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE

01583153 2000242816
Angiotensin II induces leukocyte-endothelial cell interactions in vivo via ATinf 1 and ATinf 2 receptor-mediated P-selectin upregulation
Piqueras L.; Kubes P.; Alvarez A.; O'Connor E.; Issekutz A.C.; Esplugues J.V.; Sanz M.-J.
ADDRESS: Dr. M.-J. Sanz, Departamento de Farmacologia, Facultad de Medicina, Av. Blasco Ibanez, 15-17, 46010 Valencia, Spain
EMAIL: maria.j.sanz@uv.es
Journal: Circulation, 102/17 (2118-2123), 2000, United States
PUBLICATION DATE: October 24, 2000
CODEN: CIRCA
ISSN: 0009-7322
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 26

6/3/39 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

0078325048 EMBASE No: 2000374651
Angiotensin II induces leukocyte-endothelial cell interactions in vivo via AT SUB 1 and AT SUB 2 receptor-mediated P-selectin upregulation
Piqueras L.; Kubes P.; Alvarez A.; O'Connor E.; Issekutz A.C.; Esplugues J.V.; Sanz M.-J.
Departamento de Farmacologia, Facultad de Medicina, Av. Blasco Ibanez, 15-17, 46010 Valencia, Spain
CORRESP. AUTHOR/AFFIL: Sanz M.-J.: Departamento de Farmacologia, Facultad de Medicina, Av. Blasco Ibanez, 15-17, 46010 Valencia, Spain
CORRESP. AUTHOR EMAIL: maria.j.sanz@uv.es

Circulation (Circulation) (United States) October 24, 2000, 102/17 (2118-2123)
CODEN: CIRCA ISSN: 0009-7322
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 26

6/3/40 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

0077988152 EMBASE No: 2000037327
Role of transforming growth factor-betal in cardiovascular inflammatory changes induced by chronic inhibition of nitric oxide synthesis

Koyanagi M.; Egashira K.; Kubo-Inoue M.; Usui M.; Kitamoto S.; Tomita H.; Shimokawa H.; Takeshita A.
Dept. of Cardiovascular Medicine, Cardiovascular Science, Kyushu University, Fukuoka, Japan
AUTHOR EMAIL: egashira@cardio1.med.kyushu-u.ac.jp
CORRESP. AUTHOR/AFFIL: Egashira K.: Dept. of Cardiovascular Medicine, Graduate School of Medical Sciences, Kyushu University, 3-1-1, Maidashi, Higashi-ku, Fukuoka 812-8582, Japan
CORRESP. AUTHOR EMAIL: egashira@cardio1.med.kyushu-u.ac.jp

Hypertension (Hypertension) (United States) January 1, 2000, 35/1 I (86-90)
CODEN: HPRTD ISSN: 0194-911X
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 29

6/3/41 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2008 Elsevier B.V. All rts. reserv.

0076878305 EMBASE No: 1997171390
Soluble P-selectin in hyperlipidaemia with and without symptomatic vascular disease: Relationship with von Willebrand factor
Blann A.D.; Goode G.K.; Miller J.P.; McCollum C.N.
Thromb. Haemostasis Vasc. Biol. U., University Department of Medicine, City Hospital, Birmingham, United Kingdom; Thromb. Haemostasia Vasc. Biol. U., University Department of Medicine, City Hospital, Dudley Road, Birmingham B18 7QH, United Kingdom
CORRESP. AUTHOR/AFFIL: Blann A.D.: THVBU, University Department of Medicine, City Hospital, Dudley Road, Birmingham B18 7QH, United Kingdom

Blood Coagulation and Fibrinolysis (BLOOD COAGUL. FIBRINOLYSIS) (United Kingdom) June 24, 1997, 8/3 (200-204)
CODEN: BLFIE ISSN: 0957-5235
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 29

6/3/42 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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13602692 PMID: 10833791
[Soluble P-selectin - a marker of platelet activation and vessel wall injury: increase of soluble P-selectin in plasma of patients with myocardial infarction, massive atherosclerosis and primary pulmonary hypertension]
Rastvorimui P-selektin - marker aktivatsii trombotsitov i porazheniia sosudistoi stenki: povyshenie ego urovnia v plazme krovi pri infarkte miokarda, rasprostranennom ateroskleroze i pervichnoi legochnoi gipertonii.
Semenov A V; Kogan-Ponomarev M Ia; Ruda M Ia; Komarov A L; Panchenko E P; Chazova I E; Mazurov A V
Terapevticheskii arkhiv (RUSSIA) 2000, 72 (4) p15-20, ISSN 0040-3660--Print Journal Code: 2984818R
Publishing Model Print
Document type: Comparative Study; English Abstract; Journal Article
Languages: RUSSIAN
Main Citation Owner: NLM

Record type: MEDLINE; Completed

6/3/43 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2008 Dialog. All rts. reserv.

12666370 PMID: 9588073
[Vascular endothelium as a factor in information transfer between the cardiovascular and immune systems]
Cievny endotel ako operator prenosu informacii medzi kardiovaskularnym a imunitnym systemom.
Stvrtinova V; Ferencik M; Hulin I; Jahnova E
II. interna klinika Lekarskej fakulty Univerzity Komenskeho v Bratislave.
Bratislavske lekarske listy (SLOVAKIA) Jan 1998, 99 (1) p5-19,
ISSN 0006-9248--Print Journal Code: 0065324
Publishing Model Print
Document type: English Abstract; Journal Article; Review
Languages: SLOVAK
Main Citation Owner: NLM
Record type: MEDLINE; Completed

6/3/44 (Item 1 from file: 370)
DIALOG(R)File 370:Science
(c) 1999 AAAS. All rts. reserv.

00500312 (USE 9 FOR FULLTEXT)
Molecular Therapies for Vascular Diseases
Gibbons, Gary H.; Dzau, Victor J.
The authors are at the Falk Cardiovascular Research Center, Stanford University School of Medicine, Stanford, CA 94305-5246, USA.
Science Vol. 272 5262 pp. 689
Publication Date: 5-03-1996 (960503) Publication Year: 1996
Document Type: Journal ISSN: 0036-8075
Language: English
Section Heading: Articles
Word Count: 3885

6/3/45 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2008 American Chemical Society. All rts. reserv.

130033498 CA: 130(4)33498z PATENT
Methods for preventing progressive tissue necrosis, reperfusion injury, bacterial translocation and adult respiratory distress syndrome by using dehydroepiandrosterone derivatives
INVENTOR(AUTHOR): Araneo, Barbara A.; Daynes, Raymond A.; Orlinska, Urszula; Farrukh, Imad S.
LOCATION: USA
ASSIGNEE: University of Utah Research Foundation; Pharmadigm, Inc.
PATENT: PCT International ; WO 9855074 A2 DATE: 19981210
APPLICATION: WO 98US11141 (19980603) *US 870234 (19970605)
PAGES: 55 pp. CODEN: PIXXD2 LANGUAGE: English
PATENT CLASSIFICATIONS:
CLASS: A61K-000/A
DESIGNATED COUNTRIES: AU; CA; JP DESIGNATED REGIONAL: AT; BE; CH; CY; DE ; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE
? t s6/7/all
>>>Format 7 is not valid in file 143

6/7/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15934018 BIOSIS NO.: 200100105857
Selectins level may be useful clinical markers for endothelial damage in hypertension
AUTHOR: Sanada Hironobu (Reprint); Midorikawa Sanae (Reprint); Hashimoto Shigeatsu (Reprint); Ohashi Hiroko (Reprint); Watanabe Hidetsuna; Hayashi Yoshimitsu; Katoh Tetsuo; Watanabe Tsuyoshi
AUTHOR ADDRESS: Fukushima Medical Univ, Fukushima, Japan**Japan
JOURNAL: Circulation 102 (18 Supplement): pII.321 October 31, 2000 2000
MEDIUM: print
CONFERENCE/MEETING: Abstracts from American Heart Association Scientific Sessions 2000 New Orleans, Louisiana, USA November 12-15, 2000; 20001112
SPONSOR: American Heart Association
ISSN: 0009-7322
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

6/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15933873 BIOSIS NO.: 200100105712
Markers of haemostasis and risk stratification in hypertension using the Framingham equation
AUTHOR: Spencer C (Reprint); Gurney D (Reprint); Blann A (Reprint); Beevers D (Reprint); Lip G (Reprint)
AUTHOR ADDRESS: Haemostasis, Thrombosis and Vascular Biology Unit, University Department of Medecine, City Hospital, Birmingham, UK**UK
JOURNAL: Journal of Hypertension 18 (Suppl. 4): pS149-S150 2000 ***2000***
MEDIUM: print
CONFERENCE/MEETING: 18th Scientific Meeting of the International Society of Hypertension Chicago, Illinois, USA August 20-24, 2000; 20000820
SPONSOR: International Society of Hypertension
ISSN: 0263-6352
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

6/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15804991 BIOSIS NO.: 200000523304
Angiotensin II induces leukocyte-endothelial cell interactions in vivo via AT1 and AT2 receptor-mediated P-selectin upregulation
AUTHOR: Piqueras Laura; Kubes Paul; Alvarez Angeles; O'Connor Enrique; Issekutz Andrew C; Esplugues Juan V; Sanz Maria-Jesus (Reprint)
AUTHOR ADDRESS: Departamento de Farmacologia, Facultad de Medicina, Av. Blasco Ibanez, 15-17, 46010, Valencia, Spain**Spain
JOURNAL: Circulation 102 (17): p2118-2123 October 24, 2000 2000
MEDIUM: print
ISSN: 0009-7322
DOCUMENT TYPE: Article

RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background-Angiotensin II (Ang II) plays a critical role in the development of vascular lesions in hypertension, ***atherosclerosis*** , and several renal diseases. Because Ang II may contribute to the leukocyte recruitment associated with these pathological states, the aim of the present study was to assess the role of Ang II in leukocyte-endothelial cell interactions in vivo. Methods and Results-Intravital microscopy of the rat mesenteric postcapillary venules was used. Sixty minutes of superfusion with 1 nmol/L Ang II induced a significant increase in leukocyte rolling flux (83.8 +- 20.7 versus 16.4 +- 3.1 cells/min), adhesion (11.4 +- 1.0 versus 0.8 +- 0.5 cells/100 mum), and emigration (4.0 +- 0.7 versus 0.2 +- 0.2 cells/field) without any vasoconstrictor activity. These effects were not mediated by mast cell activation. Intravenous pretreatment with AT1 (losartan) or AT2 (PD123,319) receptor antagonists significantly reduced Ang II-induced responses. A combination of both receptor antagonists inhibited the leukocyte rolling flux, adhesion, and extravasation elicited by Ang II at 60 minutes. Pretreatment of animals with fucoidin or an adhesion-blocking anti-rat P-selectin monoclonal antibody abolished Ang II-induced leukocyte responses. Furthermore, rat platelet ***P*** - ***selectin*** expression was not affected by Ang II stimulation. Conclusions-Ang II induces significant leukocyte rolling, adhesion, and emigration, which may contribute not only to hypertension but also to the onset and progression of the vascular damage associated with disease states in which plasma levels of this peptide are elevated.

6/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15603886 BIOSIS NO.: 200000322199
Soluble P-selectin: A marker of platelet activation and vessel wall injury. Increase of soluble ***P*** - ***selectin*** in the blood plasma of patients with myocardial infarction, massive atherosclerosis and primary pulmonary hypertension
AUTHOR: Semenov A V; Kagan-Ponomarev M Ya; Ruda M Ya; Komarov A L; Panchenko E P; Chazova I E; Mazurov A V
JOURNAL: Terapevticheskii Arkhiv 72 (4): p15-20 2000 2000
MEDIUM: print
ISSN: 0040-3660
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: Russian

ABSTRACT: Aim: A comparative analysis of the content of the soluble form of cell adhesion protein P-selectin in the blood plasma of patients with acute myocardial infarction (AMI), massive atherosclerosis (MA) and primary pulmonary hypertension (PPH), investigation of the relationship between plasma content of P-selectin and known markers of platelets and endothelial cells activation, preliminary assessment of the prognostic value of ***P*** - ***selectin*** determination. Materials and methods: This study included 16 patients with AMI, 20 patients with MA, 21 patients with PPH and 18 healthy donors. The follow-up was 1-5 years. End-points in the group of patients with AMI were recurrent acute coronary syndrome and coronary artery by-pass operation, in the group with MA - thrombotic complications (acute coronary syndrome, ischemic stroke) and in the group with PPH - death. ***P*** - ***selectin*** was measured by ELISA and

platelet factor 4 (PF4), thromboxane B2 (TXB2), endothelin-1 and stable prostacyclin metabolite 6-keto-prostaglandin F1alpha(6-keto-PGF1alpha) by means of commercial ELISA kits. Results: Mean level of ***P*** - selectin in blood plasma of patients with AMI (1 day) (361+-18 ng/ml), MA (410+-31 ng/ml) and PPH (627+-83 ng/ml) was increased in comparison with the group of healthy donors (269+-12 ng/ml) (everywhere p<0.001). In AMI, ***P*** - ***selectin*** was increased on day 1 only, on days 2, 3 and 10-14 of the disease the level of P-selectin was significantly lower than on day 1 and did not differ from the control level in the group of donors. In patients with MA a significant correlation was detected between plasma content of P-selectin and platelet activation marker PF4 (r=0.606, P=0.007) and in patients with PPH between the content of P-selectin and another platelet activation marker TXB2 (r=0.622, p=0.013). However, no correlation was found in PPH patients between the content of P-selectin and markers of endothelial activation and/or damage (endothelin-1 and 6-keto-PGF1alpha). Difference in the concentration of P-selectin in patients with or without end-points during the follow-up period was detected in patients with AMI (353+-14 ng/ml and 451+-24 ng/ml, p=0.009) and PPH (477+-58 ng/ml and 927+-184 ng/ml, p=0.017) but not with MA (426+-37 ng/ml and 361+-24 ng/ml, p=0.295). Conclusion: The level of P-selectin in plasma was increased in patients with acute thrombosis (AMI, 1 day) as well as in patients without clinical signs of thrombosis but with a massive injury of the vasculature (MA and PPH). The increase of ***P*** - ***selectin*** was, presumably, caused by its secretion from activated platelets since its concentration in plasma correlated with platelet concentration but not endothelial activation markers. Preliminary data indicate that blood plasma soluble P-selectin may be considered as a potential prognostic marker in AMI and PPH.

15593685 BIOSIS NO.: 200000311998
Endothelial function and hemostasis
AUTHOR: Becker B F (Reprint); Heindl B; Kupatt C; Zahler S
AUTHOR ADDRESS: Dept. of Physiology, University of Munich, Pettenkofer Str. 12, D-80336, Munich, Germany**Germany
JOURNAL: Zeitschrift fuer Kardiologie 89 (3): p160-167 March, 2000
2000
MEDIUM: print
ISSN: 0300-5860
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The vascular endothelium influences not only the three classically interacting components of hemostasis: the vessel, the blood platelets and the clotting and fibrinolytic systems of plasma, but also the natural sequelae: inflammation and tissue repair. Two principal modes of endothelial behaviour may be differentiated, best defined as an anti- and a prothrombotic state. Under physiological conditions endothelium mediates vascular dilatation (formation of NO, PGI2, adenosine, hyperpolarising factor), prevents platelet adhesion and activation (production of adenosine, NO and PGI2, removal of ADP), blocks thrombin formation (tissue factor pathway inhibitor, activation of protein C via thrombomodulin, activation of antithrombin III) and mitigates fibrin deposition (t- and scuplasminogen activator production). Adhesion and

transmigration of inflammatory leukocytes are attenuated, e.g. by NO and IL-10, and oxygen radicals are efficiently scavenged (urate, NO, glutathione, SOD). When the endothelium is physically disrupted or functionally perturbed by postischemic reperfusion, acute and chronic inflammation, atherosclerosis, diabetes and chronic arterial ***hypertension*** , then completely opposing actions pertain. This prothrombotic, proinflammatory state is characterised by vasoconstriction, platelet and leukocyte activation and adhesion (externalisation, expression and upregulation of von Willebrand factor, platelet activating factor, P-selectin, ICAM-1, IL-8, MCP-1, TNFalpha, etc.), promotion of thrombin formation, coagulation and fibrin deposition at the vascular wall (expression of tissue factor, PAI-1, phosphatidyl serine, etc.) and, in platelet-leukocyte coaggregates, additional inflammatory interactions via attachment of platelet CD40-ligand to endothelial, monocyte and B-cell CD40. Since thrombin formation and inflammatory stimulation set the stage for later tissue repair, complete abolition of such endothelial responses cannot be the goal of clinical interventions aimed at limiting procoagulatory, prothrombotic actions of a dysfunctional vascular endothelium.

6/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15565051 BIOSIS NO.: 200000283364
Vascular endothelial cell activation associated with increased plasma asymmetric dimethyl arginine in children and young adults with hypertension: A basis for atheroma?
AUTHOR: Goonasekera Chulananda D A; Shah Vanita; Rees D D; Dillon Michael J (Reprint)
AUTHOR ADDRESS: Nephrourology Unit, Institute of Child Health, 30 Guilford Street, London, WC1 N1EH, UK**UK
JOURNAL: Blood Pressure 9 (1): p16-21 2000 2000
MEDIUM: print
ISSN: 0803-7051
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The mechanism behind the development of vascular complications of ***hypertension*** in the young human remains unclear. To explore the role of vascular endothelium-generated nitric oxide (a known mediator of leucocyte-platelet-endothelial interactions) in this context, we investigated markers of endothelial activation (soluble vascular cell adhesion molecule-1 (VCAM-1), intercellular adhesion molecule-1 (ICAM-1), P-selectin, E-selectin), and von Willebrand factor and the plasma level of the endogenous nitric oxide inhibitor asymmetric dimethyl arginine (ADMA) in a group of 31 (17 male, mean age 9.4 years) ***hypertensive*** and 9 (4 male, mean age 9.1 years) healthy, normotensive children and young adults. We found raised levels of ADMA (mean (SEM) 235 (32) n mol/l) and VCAM-1 (median (range) 1237 (675-2700) ng/ml) in the plasma of hypertensive subjects compared with those of normotensives (ADMA, 103 (7) n mol/l and VCAM-1, 1005 (425-1650) ng/ml, respectively). Furthermore, in ***hypertensive*** subjects, higher VCAM-1 concentrations ($r = 0.66$, $p < 0.001$) and vWF concentrations ($r = 0.37$, $p = 0.04$) were significantly associated with a higher plasma ADMA level. Therefore, an isolated increase in plasma VCAM-1 in hypertensives in association with raised ADMA may signify a selective "non-inflammatory" endothelial activation triggered by endothelial nitric oxide synthase inhibition. Since VCAM-1 is implicated

in the origins of atherosclerosis, ADMA may be an important contributory factor in increasing the risk of atheroma formation in ***hypertensive*** children and young adults.

6/7/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15341208 BIOSIS NO.: 200000059521
Endothelin-1 causes P-selectin-dependent leukocyte rolling and adhesion within rat mesenteric microvessels
AUTHOR: Sanz Maria-Jesus (Reprint); Johnston Brent; Issekutz Andrew; Kubes Paul
AUTHOR ADDRESS: Departamento de Farmacologia, Facultat de Medicina, Universitat de Valencia, Av. Blasco Ibanez, 15-17, 46010, Valencia, Spain **Spain
JOURNAL: American Journal of Physiology 277 (5 PART 2): pH1823-H1830 Nov., 1999 1999
MEDIUM: print
ISSN: 0002-9513
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Endothelin-1 (ET-1) is a potent vasoconstrictor postulated to play a role in hypertension, ischemia-reperfusion, and ***atherosclerosis*** . In addition to these contributions, it has been also proposed to induce leukocyte-endothelial cell interactions. The aim of the present study was to assess the mechanisms of action of ET-1 on leukocyte recruitment in vivo. Intravital microscopy of the rat mesenteric postcapillary venules was used. Ten minutes after 1 nM ET-1 superfusion, a significant increase in leukocyte rolling (77.5 +- 22.6 vs. 20.5 +- 4.5 cells/min) and adhesion (15.5 +- 2.9 vs. 3.0 +- 0.8 cells/100 mum) but not emigration was observed. These effects were found not to be mediated by mast cell activation. No platelet-endothelial cell interactions were detected in this in vivo system and furthermore, flow cytometry analysis revealed no increase of P-selectin expression in rat platelets on ET-1 stimulation. Pretreatment of animals with an anti-rat P-selectin monoclonal antibody (mAb) dramatically reduced leukocyte rolling and adhesion by 100 and 94% respectively when compared with control mAb-treated animals. At this dose of ET-1, a very transient decrease in shear rate was detected, arteriolar diameter was significantly reduced but venular diameter remained unchanged. A similar mechanical reduction in blood flow did not induce leukocyte recruitment. Thus this study demonstrates that ET-1 can directly cause significant leukocyte rolling and adhesion adding to its potential pathophysiological role in the development of disease states of the cardiovascular system.

6/7/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14918499 BIOSIS NO.: 199900178159
Levels of soluble cell adhesion molecules in patients with angiographically defined coronary atherosclerosis
AUTHOR: Saku Keijiro (Reprint); Zhang Bo; Ohta Takao; Shirai Kazuyuki; Tsuchiya Yoshihiro; Arakawa Kikuo
AUTHOR ADDRESS: Department of Internal Medicine, Fukuoka University School

of Medicine, 7-45-1 Nanakuma Jonan-ku, Fukuoka, 814-0180, Japan**Japan
JOURNAL: Japanese Circulation Journal 63 (1): p19-24 Jan., 1999 ***1999***
MEDIUM: print
ISSN: 0047-1828
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Adhesion molecules on the endothelial cell membrane play an important role in the pathogenesis of ***atherosclerosis*** . Levels of soluble forms of cell adhesion molecules are reportedly elevated in patients with peripheral artery vessel disease and in patients with an atherosclerotic aorta. The present study investigated the association of serum levels of soluble vascular cell adhesion molecule 1 (sVCAM-1), soluble intercellular adhesion molecule 1 (sICAM-1), and soluble P-selectin (sP-selectin) with coronary heart disease (CHD) and the extent of coronary atherosclerosis, and examined the influence of serum levels of lipids, lipoproteins and apolipoproteins (apo) in subjects with (n=52, M/F: 43/9) and without (controls, n=40, M/F:25/15) angiographically proven coronary ***atherosclerosis*** . After controlling for age and gender, levels of sVCAM-1 (least squares mean+-std error: 565+-36 ng/ml vs 540+-41 ng/ml, ns), sICAM-1 (261+-17 ng/ml vs 247+-19 ng/ml, ns), and sP-selectin (142+-8 ng/ml vs 149+-10 ng/ml, ns) in patients with coronary atherosclerosis were not different from those in controls, as assessed by an analysis of covariance. After also adjusting for body mass index, hypertension, diabetes mellitus, and smoking by a multiple logistic function analysis, the association of sVCAM-1, sICAM-1, and sP-selectin with CHD was still not significant. Levels of sVCAM-1, sICAM-1, and sP-selectin were also not related to the extent of coronary atherosclerosis as judged by the number of stenosed vessels. However, inverse ($p<0.05$) relationships were observed between sVCAMs and serum levels of HDL3-cholesterol, apo A-II, and lipoprotein containing apo A-I and A-II, between sICAMs and levels of apo A-II and Lp A-I/A-II (Lp A-I/A-II), and between sP-selectin and lipoprotein containing only apo A-I. In conclusion, serum levels of soluble VCAM-1, ICAM-1, and P-selectin were not related to CHD or the extent of coronary atherosclerosis, but were inversely related to serum levels of high-density lipoprotein-related lipoproteins.

6/7/9 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14278298 BIOSIS NO.: 199800072545
Relation of endothelium, thrombogenesis, and hemorheology in systemic hypertension to ethnicity and left ventricular hypertrophy
AUTHOR: Lip Gregory Y H (Reprint); Blann Andrew D; Jones Alan F; Lip Peck Lin; Beevers D Gareth
AUTHOR ADDRESS: Haemostasis Thrombosis and Vasc. Biol. Unit, Univ. Dept. Med., City Hosp., Birmingham B18 7QH, UK**UK
JOURNAL: American Journal of Cardiology 80 (12): p1566-1571 Dec. 15, 1997 1997
MEDIUM: print
ISSN: 0002-9149
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Although the arterial tree is exposed to increased pressure in hypertensive patients, paradoxically, the complications of

hypertension (heart attacks, stroke) are mainly thrombotic rather than hemorrhagic. Patients with left ventricular (LV) hypertrophy are at high risk of the complications of ***hypertension*** . We performed a cross-sectional study of 178 patients attending a hypertension clinic in a city center teaching hospital, and measured plasma levels of the soluble adhesion molecule P-selectin (associated with platelet activity/function and atherosclerosis), the von Willebrand factor (vWf; a marker of endothelial dysfunction), fibrin D-dimer (an index of thrombogenesis), plasminogen activator inhibitor (PAI, an index of fibrinolysis), lipoprotein(a) (Lp(a), associated with thrombogenesis and atherogenesis) and hemorheological indexes (fibrinogen, hematocrit, plasma viscosity, hemoglobin) in patients with essential hypertension, in whom the LV mass and LV mass index were determined using echocardiography. The 178 patients (86 men, mean age 54 +- 15 years) were compared with 47 normotensive healthy controls (aged 56 +- 20 years). ***Hypertensive*** patients had higher ***P*** - ***selectin*** , PAI, vWf, fibrin D-dimer, Lp(a), plasma fibrinogen, and plasma viscosity when compared with controls. Black ***hypertensive*** patients had higher Lp(a) levels and LV septal and posterior wall thickness on echocardiography, but lower plasma PAI levels. Patients with LV hypertrophy (defined as a LV mass index > 134 g/m2 in men or > 110 g/m2 in women) had higher plasma fibrinogen compared with those without LV hypertrophy. Systolic blood pressures were significantly correlated to age, plasma viscosity, plasma fibrinogen, and vWf. Diastolic blood pressures were significantly correlated with age and plasma fibrinogen. Fibrinogen levels were correlated with LV mass, LV mass index, left atrial size, plasma viscosity, and vWf. Fibrin D-dimer levels were significantly correlated with vWf and fibrinogen levels. Thus, hypertensive patients have high plasma fibrinogen levels, thrombogenesis, and impaired fibrinolysis (as indicated by high D-dimer and PAI levels, respectively), platelet activation (raised soluble ***P*** - ***selectin***), and endothelial dysfunction (high vWF). The high plasma fibrinogen levels were related to blood pressures, LV mass index (and LV hypertrophy), and left atrial size. These abnormalities in hemorheologic factors and markers of thrombogenesis and endothelial function may act synergistically to increase the risk of thrombogenesis and ***atherosclerosis*** in ***hypertensive*** patients.

6/7/10 (Item 10 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13834186 BIOSIS NO.: 199799468246
Circulating vascular cell adhesion molecule-1 correlates with the extent of human atherosclerosis in contrast to circulating intercellular adhesion molecule-1, E-selectin, P-selectin, and thrombomodulin
AUTHOR: Peter Karlheinz (Reprint); Nawroth Peter; Conradt Christian; Nordt Thomas; Weiss Thomas; Boehme Michael; Wunsch Andreas; Allenberg Jens; Kuebler Wolfgang; Bode Christoph
AUTHOR ADDRESS: Internal Med. III, Univ. Heidelberg, Bergheimer Strasse 58, 69115 Heidelberg, Germany**Germany
JOURNAL: Arteriosclerosis Thrombosis and Vascular Biology 17 (3): p505-512 1997 1997
ISSN: 1079-5642
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Secondary prevention of atherosclerosis, especially before

the onset of symptoms, appears desirable and could be possible with a serum marker detecting ***atherosclerosis*** . Circulating, shedded forms of adhesion molecules may serve as such because their expression is upregulated in atherosclerotic plaques. In 52 patients with peripheral arterial vascular disease (Fontaine class IIa, 7 patients; class IIb, 29 patients; and class III, 16 patients), the extent of atherosclerosis was evaluated on the basis of angiograms of a large portion of the arterial system. The area diseased by atherosclerosis was determined by the percentage of vessel wall irregularities of the following calculated segments: aorta (distal from the kidney arteries), common iliac artery, external iliac artery, common femoral artery, lateral circumflex femoral artery, and popliteal artery. The maximal surface area that could exhibit atherosclerotic changes was 250 cm-2. The serum concentration of circulating vascular cell adhesion molecule-1 (VCAM-1) correlated with the extent of atherosclerosis (r=.8, P lt .001). In contrast, circulating intercellular adhesion molecule-1, E-selectin, P-selectin, and thrombomodulin (as markers for endothelial cell damage) did not correlate with the extent of ***atherosclerosis*** . Furthermore, circulating VCAM-1 could be used to indicate stages of atherosclerosis with a high degree of statistical significance. The potential bias of factors such as age, diabetes mellitus, hypercholesterolemia, arterial hypertension, renal failure, and history of myocardial infarction on the correlation of circulating VCAM-1 with the extent of atherosclerosis could be excluded by multivariate analysis. These findings suggest an important role of VCAM-1 in atherosclerosis and may serve as the basis for further evaluation of circulating VCAM-1 as a potential serum marker for ***atherosclerosis*** .

6/7/11 (Item 11 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13207437 BIOSIS NO.: 199698675270
Soluble adhesion molecule P-selectin and endothelial dysfunction in essential hypertension: Implications for atherogenesis? A preliminary report
AUTHOR: Lip Gregory Y H (Reprint); Blann Andrew D; Zarifis John; Beevers Michele; Lip Peck-Lin; Beevers D Gareth
AUTHOR ADDRESS: Univ. Dep. Medicine, City Hosp., Dudley Rd., Birmingham B18 7QH, UK**UK
JOURNAL: Journal of Hypertension 13 (12 PART 2): p1674-1678 1995
1995
ISSN: 0263-6352
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Objective: Patients with essential hypertension are at high risk of atherosclerotic vascular disease. To investigate this further, we measured levels of the soluble adhesion molecule P-selectin, which is associated with platelet activity/function and atherosclerosis, von Willebrand factor, which is a marker of endothelial dysfunction, and plasma fibrinogen. Patients and methods: We studied 104 consecutive patients (47 males, 57 females; mean +- SD age 54.8 +- 14.1 years) with essential ***hypertension*** compared with 47 normotensive healthy controls (55.0 +- 19.2 years). Levels of soluble adhesion molecule P-selectin and von Willebrand factor were measured by enzyme-linked immunosorbent assay, and plasma fibrinogen by a clotting method (CLAUSS). Results: Compared with normotensives, the

hypertensives showed significant increases in soluble P-selectin (300 versus 228 ng/ml; median difference 55 ng/ml, Mann-Whitney test P=0.03), von Willebrand factor (114 versus 96 IU/l; unpaired t-test P ltoreq 0.001) and fibrinogen (3.3 versus 2.9 g/l; unpaired t-test P ltoreq 0.001). There were significant correlations between fibrinogen and ***P*** - ***selectin*** (r=0.16; P=0.02) and von Willebrand factor (r=0.39; P lt 0.001), but not between ***P*** - ***selectin*** and von Willebrand factor. There were no differences in these factors between patients with (n = 53) and without (n = 51) antihypertensive therapy or between those with good blood pressure control (systolic/diastolic ltoreq 160/90 mmHg; n = 17) and those with poor control. A stepwise multiple regression analysis showed that diastolic blood pressure was a significant predictor for soluble P-selectin levels; diastolic blood pressure and von Willebrand factor levels were significant predictors for fibrinogen levels (P lt 0.05). Conclusions: This study suggests that hypertensives have high plasma fibrinogen levels, platelet dysfunction (which could contribute to atherogenesis, as indicated by raised soluble P-selectin levels) and endothelial dysfunction (as indicated by high von Willebrand factor levels), which are related to diastolic blood pressure. These factors may act synergistically to increase atherogenesis and may explain the high risk of atherosclerotic vascular disease in ***hypertensives*** .

6/7/12 (Item 12 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12934590 BIOSIS NO.: 199598402423
Platelet-derived microparticles may influence the development of atherosclerosis in diabetes mellitus
AUTHOR: Nomura Shosaku (Reprint); Suzuki Masahiko; Katsura Kaoruko; Xie Gui Lan; Miyazaki Yasuhiko; Miyake Tetsuya; Kido Hirofumi; Kagawa Hideo; Fukuhara Shirou
AUTHOR ADDRESS: The First Dep. Internal Med., Kansai Med. Univ., 10-15, Fumizono-cho, Moriguchi, Osaka 570, Japan**Japan
JOURNAL: Atherosclerosis 116 (2): p235-240 1995 1995
ISSN: 0021-9150
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: We investigated the association between low-density lipoprotein (LDL), triglycerides, and platelet activation in 18 patients with hypertension age 41-64 years and 18 with diabetes mellitus aged 43-70 years. Platelet ***P*** - ***selectin*** positivity and the microparticle level (indicators of activation) were both significantly higher in the diabetics than in healthy controls (P-selectin: 28.0% +- 7.5% vs. 7.3% +- 4.2%, P lt 0.001; microparticles: 1900 +- 966 vs. 526 +- 158/10-4 platelets, P lt 0.01). In contrast, there was no significant increase of either parameter in the patients with ***hypertension*** . Plasma microparticle levels were also significantly greater in the diabetics with high LDL levels than in those with low LDL levels (2375 + 949 vs. 1519 + 796/10-4 platelets, P lt 0.05), and in those with high rather than low triglyceride levels (2188 + 845 vs. 1492 + 783/10-4 platelets, P lt 0.05). However, platelet positivity for P-selectin was not significantly different between these two subgroups. Microparticle and ***P*** - ***selectin*** levels both showed no significant difference between the hypertensive patients with high and low LDL or triglyceride levels. These results suggest that

platelet-derived microparticles may participate in the development or progression of ***atherosclerosis*** in patients with diabetes mellitus.

6/7/13 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

09085737 Genuine Article#: 365XG Number of References: 26
Title: Angiotensin II induces leukocyte-endothelial cell interactions in vivo via AT(1) and AT(2) receptor-mediated P-selectin upregulation
Author(s): Piqueras L; Kubes P; Alvarez A; OConnor E; Issekutz AC; Esplugues JV; Sanz MJ (REPRINT)
Corporate Source: UNIV VALENCIA,FAC MED, DEPT FARMACOL, AV BLASCO IBANEZ 15-17/VALENCIA 46010//SPAIN/ (REPRINT); UNIV VALENCIA,FAC MED, DEPT FARMACOL/VALENCIA 46010//SPAIN/; UNIV VALENCIA,DEPT BIOCHEM/VALENCIA 46010//SPAIN/; UNIV CALGARY,IMMUNOL RES GRP/CALGARY/AB T2N 1N4/CANADA/; DALHOUSIE UNIV,DEPT PEDIAT/HALIFAX/NS/CANADA/; DALHOUSIE UNIV,DEPT PATHOL/HALIFAX/NS/CANADA/; DALHOUSIE UNIV,DEPT MICROBIOL/HALIFAX/NS/CANADA/; DALHOUSIE UNIV,DEPT IMMUNOL/HALIFAX/NS/CANADA/
Journal: CIRCULATION, 2000, V102, N17 (OCT 24), P2118-2123
ISSN: 0009-7322 Publication date: 20001024
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621
Language: English Document Type: ARTICLE
Abstract: Background-Angiotensin II (Ang II) plays a critical role in the development of vascular lesions in hypertension, ***atherosclerosis*** , and several renal diseases. Because Ang II may contribute to the leukocyte recruitment associated with these pathological states, the aim of the present study was to assess the role of Ang II in leukocyte-endothelial cell interactions in vivo.

Methods and Results-Intravital microscopy of the rat mesenteric postcapillary venules was used. Sixty minutes of superfusion with 1 nmol/L Ang II induced a significant increase in leukocyte rolling flux (83.8+/-20.7 versus 16.4+/-3.1 cells/min), adhesion (11.4+/-1.0 versus 0.8+/-0.5 cells/100 mum), and emigration (4.0+/-0.7 versus 0.2+/-0.2 cells/field) without any vasoconstrictor activity. These effects were not mediated by mast cell activation. Intravenous pretreatment with AT(1) (losartan) or AT(2) (PD123,319) receptor antagonists significantly reduced Ang II-induced responses. A combination of both receptor antagonists inhibited the leukocyte rolling flux, adhesion, and extravasation elicited by Ang II at 60 minutes. Pretreatment of animals with fucoidin or an adhesion-blocking anti-rat P-selectin monoclonal antibody abolished Ang II-induced leukocyte responses. Furthermore, rat platelet ***P*** - ***selectin*** expression was not affected by Ang II stimulation.

Conclusions-Ang II. induces significant leukocyte rolling, adhesion, and emigration, which may contribute not only to hypertension but also to the onset and progression of the vascular damage associated with disease states in which plasma levels of this peptide are elevated.

6/7/14 (Item 2 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

08870536 Genuine Article#: 339BB Number of References: 121
Title: Testing for endothelial dysfunction
Author(s): Raitakari OT (REPRINT) ; Celermajer DS
Corporate Source: UNIV TURKU,CENT HOSP, TURKU PET CTR, POB 52/FIN-20520 TURKU//FINLAND/ (REPRINT); ROYAL PRINCE ALFRED HOSP,DEPT CARDIOL/SYDNEY/NSW/AUSTRALIA/; UNIV TURKU,DEPT CLIN PHYSIOL/TURKU//FINLAND/; UNIV TURKU,TURKU PET CTR/TURKU//FINLAND/; UNIV SYDNEY,DEPT MED/SYDNEY/NSW 2006/AUSTRALIA/
Journal: ANNALS OF MEDICINE, 2000, V32, N5 (JUL), P293-304
ISSN: 0785-3890 Publication date: 20000700
Publisher: ROYAL SOC MEDICINE PRESS LTD, 1 WIMPOLE STREET, LONDON W1M 8AE, ENGLAND
Language: English Document Type: REVIEW
Abstract: Endothelial health is a key factor in normal cardiovascular homeostasis, and recent studies have revealed several important functions of the vascular endothelium that protect against atherothrombosis. These include control over arterial tone, coagulation, fibrinolysis, and vascular growth. Consequently, endothelial dysfunction has been implicated as an important event in the pathogenesis of atherosclerosis, coronary vasoconstriction, ***hypertension*** , and myocardial ischaemia. Therefore, there has been considerable research interest in diagnostic assays for the assessment of endothelium. This review outlines the current status of markers of endothelial dysfunction, particularly those related to vasomotor control, as well as circulating markers of vascular health.

6/7/15 (Item 3 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

07920918 Genuine Article#: 224JM Number of References: 26
Title: Adhesion molecules in cerebrovascular diseases - Evidence for an inflammatory endothelial activation in cerebral large- and small-vessel disease
Author(s): Fassbender K (REPRINT) ; Bertsch T; Mielke O; Muhlhauser F; Hennerici M
Corporate Source: UNIV HEIDELBERG,KLINIKUM MANNHEIM, DEPT NEUROL, THEODOR KUTZER UFER 1-3/D-68135 MANNHEIM//GERMANY/ (REPRINT); UNIV HEIDELBERG,KLINIKUM MANNHEIM, INST CLIN CHEM/D-68135 MANNHEIM//GERMANY/
Journal: STROKE, 1999, V30, N8 (AUG), P1647-1650
ISSN: 0039-2499 Publication date: 19990800
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 227 EAST WASHINGTON SQ, PHILADELPHIA, PA 19106
Language: English Document Type: ARTICLE
Abstract: Background and Purpose-Adhesion molecules mediate attachment and transendothelial migration of leukocytes as a critical step in pathogenesis of ***atherosclerosis*** . Their expression and release were comparatively investigated in patients with large- and small-vessel disease of the central nervous system.

Methods-With immunological methods, serum concentrations of endothelial-derived adhesion molecules (soluble endothelial-leukocyte adhesion molecule [sE-selectin], soluble vascular-leukocyte adhesion molecule-1, and soluble intercellular adhesion molecule-1 [sICAM-1]) were quantified in patients with obstructive disease of extracranial (n=89) and intracranial (n=20) large-vessel disease and patients with subcortical vascular encephalopathy (n=64), a cerebral small-vessel disease. As controls, age- and sex-matched subjects without obstructive cerebrovascular disease (n=67) were studied.

Results-We observed significantly increased serum concentrations of sE-selectin and sICAM-1 in patients with both obstructive disease of the large brain-supplying arteries and subcortical vascular encephalopathy. Interestingly, the highest levels were observed in intracranial macroangiopathy. Furthermore, concentrations of sICAM-1 and sE-selectin were significantly increased in current smokers but not in diabetic or ***hypertensive*** patients.

Conclusions-The observation of elevated release of endothelial-derived adhesion molecules in both patients with stenoses of the large brain-supplying arteries and patients with subcortical vascular encephalopathy indicates that inflammatory endothelial activation and adhesion of leukocytes play similarly important roles in cerebral large- and small-vessel disease.

6/7/16 (Item 4 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

07852096 Genuine Article#: 216BG Number of References: 35
Title: Cross-sectional study of soluble intercellular adhesion molecule-1 and cardiovascular risk factors in apparently healthy men
Author(s): Rohde LEP; Hennekens CH; Ridker PM (REPRINT)
Corporate Source: BRIGHAM & WOMENS HOSP,DIV CARDIOVASC, 75 FRANCIS ST/BOSTON//MA/02115 (REPRINT); BRIGHAM & WOMENS HOSP,DIV CARDIOVASC/BOSTON//MA/02115; BRIGHAM & WOMENS HOSP,DIV PREVENT MED/BOSTON//MA/02115; HARVARD UNIV,SCH MED, DEPT AMBULATORY CARE & PREVENT/BOSTON//MA/
Journal: ARTERIOSCLEROSIS THROMBOSIS AND VASCULAR BIOLOGY, 1999, V19 , N7 (JUL), P1595-1599
ISSN: 1079-5642 Publication date: 19990700
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 227 EAST WASHINGTON SQ, PHILADELPHIA, PA 19106
Language: English Document Type: ARTICLE
Abstract: An elevated plasma concentration of the soluble intercellular adhesion molecule-1 (sICAM-1) is associated with increased risk for future coronary events. However, data exploring the interrelations of sICAM-1 with known cardiovascular risk factors are sparse. We determined sICAM-1 levels in 948 middle-aged men with no prior history of cardiovascular disease. sICAM-1 levels increased with age (P<0.001) and were significantly associated with smoking (P<0.001), ***hypertension*** (P<0.05), and frequent alcohol consumption (P=0.006). Positive correlations were observed between sICAM-1 and triglycerides (r=0.15; P<0.001), fibrinogen (r=0.21; P<0.001), tissue-type plasminogen activator antigen (r=0.17; P<0.001), and total homocysteine (r=0.09; P=0.02); whereas a negative correlation was observed for high density lipoprotein cholesterol (r=-0.15; P<0.001). Overall, plasma concentrations of sICAM-1 increased with increasing prevalence of usual cardiovascular risk factors; mean plasma concentrations were 231, 236, 245, 257, and 312 ng/mL for those subjects with 0, 1, 2, 3, and >4 risk factors, respectively (P<0.01 for trend). In multivariate analysis, age, smoking status, diabetes, systolic blood pressure, positive family history of coronary disease, and serum levels of total homocysteine and fibrinogen were all independently associated with sICAM-1 levels tall P less than or equal to 0.05). sICAM-1 levels are associated with several established cardiovascular risk factors. Further studies will be needed to evaluate whether these associations reflect the role of sICAM-1 as a marker of preclinical atherosclerosis, and whether such interrelations might have a causal basis.

6/7/17 (Item 5 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

07493240 Genuine Article#: 173CJ Number of References: 48
Title: Fosinopril decreases levels of soluble vascular cell adhesion molecule-1 in borderline hypertensive type II diabetic patients with microalbuminuria
Author(s): Gasic S (REPRINT) ; Wagner OF; Fasching P; Ludwig C; Veitl M; Kapiotis S; Jilma B
Corporate Source: UNIV HOSP VIENNA,DIV ENDOCRINOL & METAB, DEPT INTERNAL MED 3, WAHRINGERGURTEL 18-20/A-1090 VIENNA//AUSTRIA/ (REPRINT); UNIV HOSP VIENNA,DEPT CLIN PHARMACOL, ADHES RES GRP ELABORATING THERAPEUT/A-1090 VIENNA//AUSTRIA/; UNIV HOSP VIENNA,MED & CHEM LAB DIAGNOST, INST CLIN/A-1090 VIENNA//AUSTRIA/; UNIV LEIPZIG,DEPT CLIN CHEM & PATHOBIOCHEM/D-7010 LEIPZIG//GERMANY/
Journal: AMERICAN JOURNAL OF HYPERTENSION, 1999, V12, N2,1 (FEB), P 217-222
ISSN: 0895-7061 Publication date: 19990200
Publisher: ELSEVIER SCIENCE INC, 655 AVENUE OF THE AMERICAS, NEW YORK, NY 10010
Language: English Document Type: ARTICLE
Abstract: Angiotensin converting enzyme inhibitors (ACE-I) are a mainstay for the treatment of heart failure, and of diabetic microalbuminuria. Recently ACE-I have been found to decrease plasma levels of circulating vascular cell adhesion molecule-1 (cVCAM-1) in patients with congestive heart failure. As increased cVCAM-1 levels are pathognomonic for diabetics with microangiopathy, we investigated the effects of ACE-I on plasma levels of cVCAM-2 intercellular adhesion molecule (cICAM-1), and cE-selectin in microalbuminuric diabetics. In addition, the effects of ACE-I on plasma levels of plasminogen activator inhibitor (PAI-1) and of tissue plasminogen activator (TPA) were studied. Fosinopril (10 mg/day) was administered over 12 weeks to 11 microalbuminuric patients with non-insulin-dependent diabetes mellitus (NIDDM). As expected, baseline plasma concentrations of cE-selectin, cICAM-1, and cVCAM-1 were markedly higher in patients than in healthy control subjects (n = 82; P <.001). PAI-1 levels in NIDDM were similar to those in control subjects, whereas TPA levels were about 25% lower in patients than in control subjects (P =.013), Serum levels of cVCAM-1 decreased by -19% (CI: -25% to -13%) after treatment with fosinopril (P =.003) and were no longer different from those of the control group. In contrast, plasma levels of cE-selectin, cICAM-1, PAI-1, and TPA were unaffected. As expected microalbuminuria decreased by -44% (CI: -65 to -22; P =.004). In conclusion fosinopril lowered cVCAM-1 levels along with microalbuminuria in NIDDM. This may represent a novel mechanism of action of ACE-I in diabetes-associated endothelial dysfunction. Whether decreased VCAM-1 expression is responsible for the observed reduction in microalbuminuria, deserves further investigation. Am J Hypertens 1999;12:217-222 (C) 1999 American Journal of ***Hypertension*** , Ltd.

6/7/18 (Item 6 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

07309090 Genuine Article#: 145LL Number of References: 44
Title: The influence of acute smoking on leucocytes, platelets and the endothelium
Author(s): Blann AD (REPRINT) ; Kirkpatrick U; Devine C; Naser S; McCollum

CN
Corporate Source: UNIV BIRMINGHAM,CITY HOSP, DEPT MED, HAEMOSTASIS THROMBOSIS & VASC BIOL UNIT/BIRMINGHAM B18 7QH/W MIDLANDS/ENGLAND/ (REPRINT); UNIV S MANCHESTER HOSP,DEPT SURG/MANCHESTER M20 8LR/LANCS/ENGLAND/
Journal: ATHEROSCLEROSIS, 1998, V141, N1 (NOV), P133-139
ISSN: 0021-9150 Publication date: 19981100
Publisher: ELSEVIER SCI IRELAND LTD, CUSTOMER RELATIONS MANAGER, BAY 15, SHANNON INDUSTRIAL ESTATE CO, CLARE, IRELAND
Language: English Document Type: ARTICLE
Abstract: Cigarette smoking is a risk factor for the development of ***atherosclerosis*** . Possible mechanisms for this include leucocytes and platelet activation, and/or damage to the endothelium, any of which may contribute to changes in thrombosis and haemostasis. We examined the acute effects of smoking on these systems by obtaining blood before, immediately after, and at 10 and 30 min after the rapid smoking of two cigarettes in sequence by 20 smokers. Blood samples taken at the same time points from ten non-smokers acted as control material. In the smokers there was a transient rise in leucocyte count and neutrophil activation, but von Willebrand factor (VWF-marking endothelial damage) increased steadily at each time point (P < 0.05). There were no changes in neutrophil elastase, soluble intercellular adhesion molecule-1 (sICAM-1-normally increased in smokers), fibrinogen, platelet count or soluble P-selectin (marking platelet activation, also normally increased in smokers). We conclude that the acute smoking of two cigarettes in succession will activate leucocytes and cause endothelial cell damage, but will not immediately influence platelet activity. (C) 1998 Elsevier Science Ireland Ltd. All rights reserved.

6/7/19 (Item 7 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

07220837 Genuine Article#: 138QD Number of References: 36
Title: Circulating cell adhesion molecules are correlated with ultrasound-based assessment of carotid atherosclerosis
Author(s): Rohde LE; Lee RT; Rivero J; Jamacochian M; Arroyo LH; Briggs W; Rifai N; Libby P; Creager MA; Ridker PM (REPRINT)
Corporate Source: BRIGHAM & WOMENS HOSP,DIV CARDIOVASC, DEPT MED, 75 FRANCIS ST/BOSTON//MA/02115 (REPRINT); BRIGHAM & WOMENS HOSP,DIV CARDIOVASC, DEPT MED/BOSTON//MA/02115; HARVARD UNIV,SCH MED, CHILDRENS HOSP, DEPT LAB MED/BOSTON//MA/
Journal: ARTERIOSCLEROSIS THROMBOSIS AND VASCULAR BIOLOGY, 1998, V18 , N11 (NOV), P1765-1770
ISSN: 1079-5642 Publication date: 19981100
Publisher: WILLIAMS & WILKINS, 351 WEST CAMDEN ST, BALTIMORE, MD 21201-2436
Language: English Document Type: ARTICLE
Abstract: Although cellular adhesion molecules (CAMs) are hypothesized to play an important role in atherogenesis, the relationship between CAMs and systemic ***atherosclerosis*** is uncertain. Among 92 outpatients (48 men; mean+/-SD age, 65+/-9 years), we evaluated the association of soluble vascular CAM-1 (sVCAM-1) and intercellular adhesion molecule-1 (sICAM-1) with carotid intimal-medial thickness (IMT), an index of early ***atherosclerosis*** . All subjects underwent a 2-dimensional ultrasound examination of both carotid arteries at the distal common carotid arteries and bifurcation. sVCAM-1 and sICAM-1 levels measured by enzyme-linked immunosorbent assay were significantly correlated with mean IMT of the common carotid artery (r=0.34 and r=0.30, respectively; P<0.01) and carotid bifurcation (r=0.31 and r=0.26, respectively; P<0.05), whereas sVCAM-1 was also positively associated with maximal

carotid IMT (r=0.35, P<0.01). Adjustment for age attenuated the association between sVCAM-1 and common (r=0.16, P=0.13) and bifurcation (r=0.18, P=0.07) carotid IMT but had minimal effect on the associations between sICAM-1 and carotid measurements (r=0.32, P<0.01; r=0.23, P<0.05; for common and bifurcation IMT, respectively). Age-adjusted sICAM-1 levels increased in a stepwise fashion across common carotid IMT tertiles (253+/-27 versus 275+/-24 versus 384+/-26 pg/mL for the lowest, intermediate, and highest IMT tertiles, respectively; P<0.01). A similar trend was also found between sVCAM-1 levels and common carotid IMT tertiles (625+/-60 versus 650+/-53 versus 714+/-58 pg/mL; P<0.15). These associations were minimally affected in analyses adjusting for hypertension, diabetes, smoking, low and high density lipoprotein cholesterol, lipoprotein(a), and homocysteine, or in a subgroup analysis limited to those with no prior history of atherothrombotic disease. These data demonstrate a positive association between serum CAMs with carotid IMT and further support the hypothesis that systemic inflammation may have a role in atherosclerotic lesion development.

6/7/20 (Item 8 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

06430023 Genuine Article#: YT137 Number of References: 39
Title: Putative role of adhesion molecules in metabolic disorders
Author(s): Wagner OF (REPRINT) ; Jilma B
Corporate Source: UNIV LEIPZIG,DEPT CLIN CHEM & PATHOBIOCHEM, PAUL LIST STR 13/D-04103 LEIPZIG//GERMANY/ (REPRINT); UNIV HOSP VIENNA,SCH MED, ADHES RES GRP ELABORATING THERAPEUT, DEPT CLIN PHARMACOL, TARGET/VIENNA//AUSTRIA/
Journal: HORMONE AND METABOLIC RESEARCH, 1997, V29, N12 (DEC), P 627-630
ISSN: 0018-5043 Publication date: 19971200
Publisher: GEORG THIEME VERLAG, P O BOX 30 11 20, D-70451 STUTTGART, GERMANY
Language: English Document Type: ARTICLE
Abstract: Diabetes mellitus is associated with an increased risk of premature vascular disease. Vascular research is focusing on a potential role of adhesion molecules in diabetes mellitus, since classical risk factors including hyperlipidemia and hypertension do not completely account for the increased incidence of ***atherosclerosis*** in diabetes. After the expression of adhesion molecules on the cell surface, they are shed into plasma. Thus plasma concentrations of circulating adhesion molecules may be representative for endothelial activation, damage or turnover. Recently, evidence has been accumulating that increased plasma levels of adhesion molecules may predict cardiovascular disease, may be pathognomonic for diabetic microangiopathy or may even play a functional pathophysiologic role. The purpose of this article is to briefly summarise the role of (circulating) adhesion molecules in diabetes mellitus.

6/7/21 (Item 9 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

06349615 Genuine Article#: YL375 Number of References: 61
Title: Circulating adhesion molecules VCAM-1, ICAM-1, and E-selectin in carotid atherosclerosis and incident coronary heart disease cases - The atherosclerosis risk in communities (ARIC) study

Author(s): Hwang SJ; Ballantyne CM; Sharrett AR; Smith LC; Davis CE; Gotto AM; Boerwinkle E (REPRINT)
Corporate Source: UNIV TEXAS,HLTH SCI CTR, CTR HUMAN GENET, POB 20334/HOUSTON//TX/77225 (REPRINT); UNIV TEXAS,HLTH SCI CTR, CTR HUMAN GENET/HOUSTON//TX/77225; UNIV TEXAS,HLTH SCI CTR, INST MOL MED/HOUSTON//TX/77225; BAYLOR COLL MED,DEPT MED/HOUSTON//TX/77030; NHLBI,EPIDEMIOL & BIOMETRY PROGRAM/BETHESDA//MD/20892; UNIV N CAROLINA,SCH PUBL HLTH, DEPT BIOSTAT/CHAPEL HILL//NC/
Journal: CIRCULATION, 1997, V96, N12 (DEC 16), P4219-4225
ISSN: 0009-7322 Publication date: 19971216
Publisher: AMER HEART ASSOC, 7272 GREENVILLE AVENUE, DALLAS, TX 75231-4596
Language: English Document Type: ARTICLE
Abstract: Background Recruitment of circulating leukocytes at sites of atherosclerosis is mediated through a family of adhesion molecules. The function of circulating forms of these adhesion molecules remains unknown, but their levels may serve as molecular markers of subclinical coronary heart disease (CHD).

Methods and Results To determine the ability of circulating vascular cell adhesion molecule-1 (VCAM-1), endothelial-leukocyte adhesion molecule-1 (E-selectin), and intercellular adhesion molecule-1 (ICAM-1) to serve as molecular markers of atherosclerosis and predictors of incident CHD, we studied 204 patients with incident CHD, 272 patients with carotid artery atherosclerosis (CAA), and 316 control subjects from the large, biracial Atherosclerosis Risk In Communities (ARIC) study. Levels of VCAM-1 were not significantly different among the patients with incident CHD, those with CAA, and control subjects. Higher levels of E-selectin and ICAM-1 were observed for the patients with CHD (means [ng/mL]: E-selectin, 38.4; ICAM-1, 288.7) and those with CAA (E-selectin, 41.5; ICAM-1, 283.6) compared with the control subjects (E-selectin, 32.8; ICAM-1, 244.2), but the distributions were not notably different between the patients with CHD and CAA. Results of logistic regression analyses indicated that the relationship of ICAM-1 and E-selectin with CHD and CAA was independent of other known CHD risk factors and was most pronounced in the highest quartile. The odds of CHD and CAA were 5.53 (95% CI, 2.51-12.21) and 2.64 (95% CI, 1.40-5.01), respectively, for those with levels of ICAM-1 in the highest quartile compared with those in the lowest quartile. Odds of CAA were 2.03 (95% CI, 1.14-3.62) for those with levels of E-selectin in the highest quartile compared with those in the lowest quartile.

Conclusions These data indicate that plasma levels of ICAM-1 and E-selectin may serve as molecular markers for atherosclerosis and the development of CHD.

6/7/22 (Item 10 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

06221361 Genuine Article#: YC877 Number of References: 69
Title: Endothelial cell injury in cardiovascular surgery: An overview
Author(s): Verrier ED (REPRINT) ; Boyle EM
Corporate Source: UNIV WASHINGTON,DEPT SURG, DIV CARDIOTHORAC SURG, 1059 PACIFIC AVE NE, BOX 356310/SEATTLE//WA/98195 (REPRINT)
Journal: ANNALS OF THORACIC SURGERY, 1997, V64, N4,S (OCT), PS2-S8
ISSN: 0003-4975 Publication date: 19971000
Publisher: ELSEVIER SCIENCE INC, 655 AVENUE OF THE AMERICAS, NEW YORK, NY 10010
Language: English Document Type: ARTICLE
Abstract: In the last decade the endothelium has been shown to play a major

role in regulating membrane permeability, lipid transport, vasomotor tone, coagulation, inflammation, and vascular wall structure. These critical endothelial cell functions are extremely sensitive to injury in the form of hypoxia, exposure to cytokines, endotoxin, cholesterol, nicotine, surgical manipulation, or hemodynamic shear stress. In response to injury endothelial cells become activated, tipping the balance of endothelial-derived factors to disrupt barrier function, and enhance vasoconstriction, coagulation, leukocyte adhesion, and smooth muscle cell proliferation. Although these responses likely exist as protective mechanisms, if the stimuli are severe the responses may become excessive, resulting in damaged tissue, impaired organ function, and an abnormal fibroproliferative response. Recent discoveries in the field of vascular biology have led to an expanded understanding of many of the complications of cardiovascular operations. Because of the wide impact endothelial cell dysfunction has on patients with cardiovascular disease, issues pertaining to endothelial biology are in the forefront of research that will affect the current and future practice of cardiothoracic surgery. (C) 1996 by The Society of Thoracic Surgeons.

6/7/23 (Item 11 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05962307 Genuine Article#: XK575 Number of References: 37
Title: Anticardiolipin antibodies are not associated with restenosis or endothelial activation after percutaneous transluminal angioplasty
Author(s): Tsakiris DA (REPRINT) ; Tschopl M; Jager K; Wolf F; Marbet GA
Corporate Source: UNIV BASEL HOSP,HAEMOSTASIS LAB, DEPT CENT LAB/CH-4031 BASEL//SWITZERLAND/ (REPRINT)
Journal: INTERNATIONAL ANGIOLOGY, 1997, V16, N2 (JUN), P88-93
ISSN: 0392-9590 Publication date: 19970600
Publisher: EDIZIONI MINERVA MEDICA, CORSO BRAMANTE 83-85 INT JOURNALS DEPT., 10126 TURIN, ITALY
Language: English Document Type: ARTICLE
Abstract: Objective. Restenosis following percutaneous transluminal angioplasty (PTA) continues to be a major clinical problem. Anticardiolipin antibodies (aCL) have been established as risk factors for venous or arterial thrombosis. The aim of this study was to assess: a) the influence of positive aCL upon restenosis within 6 months after PTA, b) the possibility of a seroconversion from negative to positive aCL after PTA and c) a possible link between positive aCL and endothelial activation.

Experimental design. 71 patients (50 men and 21 women, age 68 +/- 13 years) with peripheral arterial occlusive disease (PAOD, Fontaine II-IV) undergoing a successful PTA entered the study and were prospectively followed for 3 and 6 months thereafter.

Interventions. PTA was carried out successfully and noninvasive grading was done with duplex scanning. Laboratory investigation included aCL, thrombin generation markers, such as thrombin-antithrombin III complexes and prothrombin fragments 1 + 2, as well as thrombomodulin, soluble P-selectin, E-selectin and the vascular cell adhesion molecule-1, as endothelial activation markers.

Results. 30/71 (42.3%) patients developed restenosis (> 50% reduction of the lumen diameter) within 6 months after PTA. 9/71 (12.7%), had positive aCL IgG (19-35 GPL) and/or IgM (14-103 MPL) at all three measurements. 2/9 (22.2%) of aCL positive and 28/62 (45.2%)

of aCL negative patients had restenosis at 6 months after PTA (relative risk RR = 0.51, 95%-CI: 0.14-1.78, chi(2) non-significant). All other parameters did not differ between aCL-positive and -negative groups.

Conclusions. Our findings suggest that: a) patients with PAOD have a slightly higher prevalence of positive aCL compared to the general population, but no association is evident between positive aCL and restenosis within 6 months after PTA, b) no seroconversion from negative to positive aCL occurred within 6 months after PTA, c) no association of aCL with endothelial activation markers or thrombin generation markers was found.

6/7/24 (Item 12 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05920616 Genuine Article#: XG541 Number of References: 104
Title: Vascular homeostasis, adhesion molecules, and macrovascular disease in non-insulin-dependent diabetes mellitus
Author(s): Carter AM; Grant PJ (REPRINT)
Corporate Source: UNIV LEEDS,RES SCH MED, UNIT MOL VASC MED, LEEDS GEN INFIRM, G FLOOR, MARTIN WING/LEEDS LS1 3EX/W YORKSHIRE/ENGLAND/ (REPRINT); UNIV LEEDS,RES SCH MED, UNIT MOL VASC MED, LEEDS GEN INFIRM/LEEDS LS1 3EX/W YORKSHIRE/ENGLAND/
Journal: DIABETIC MEDICINE, 1997, V14, N6 (JUN), P423-432
ISSN: 0742-3071 Publication date: 19970600
Publisher: JOHN WILEY & SONS LTD, BAFFINS LANE CHICHESTER, W SUSSEX, ENGLAND PO19 1UD
Language: English Document Type: REVIEW
Abstract: Diabetes mellitus is characterized by fasting hyperglycaemia and the development of chronic vascular complications. While microvascular disease has been strongly related to glycaemic control, the major cause of mortality in diabetes is due to macrovascular disease affecting the cardiac and cerebrovascular circulations, which appear to have a more complex pathogenesis. Diabetes is associated with a 3-5-fold increase in death from myocardial infarction and similar figures pertain to stroke. The processes involved in atherothrombotic disease are complex and include variation in lipid metabolism, vascular responses, cell/cell interactions, and in the fluid and cellular phases of coagulation and fibrinolysis. The complex interactions between all of these processes are crucially altered by the metabolic milieu that characterizes diabetes mellitus, tipping the delicate balance towards atheroma formation, platelet aggregation and thrombus formation. This article will review these mechanisms and the effects of diabetes in the pathogenesis of vascular disease. (C) 1997 by John Wiley & Sons, Ltd.

6/7/25 (Item 13 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05847709 Genuine Article#: XB411 Number of References: 38
Title: Leukocyte activation in atherosclerosis: Correlation with risk factors
Author(s): Elneihoum AM (REPRINT) ; Falke P; Hedblad B; Lindgarde F; Ohlsson K
Corporate Source: UNIV LUND HOSP,DEPT MED/S-20502 MALMO//SWEDEN/ (REPRINT); UNIV LUND HOSP,DEPT SURG PATHOPHYSIOL/S-20502 MALMO//SWEDEN/
Journal: ATHEROSCLEROSIS, 1997, V131, N1 (MAY), P79-84
ISSN: 0021-9150 Publication date: 19970500

Publisher: ELSEVIER SCI IRELAND LTD, CUSTOMER RELATIONS MANAGER, BAY 15, SHANNON INDUSTRIAL ESTATE CO, CLARE, IRELAND
Language: English Document Type: ARTICLE
Abstract: Leukocytes have been implicated in the development of atherosclerotic vascular diseases, and numerous abnormalities of leukocytes in conjunction with atherosclerosis have been reported. The aim of this study of middle-aged asymptomatic subjects with early atherosclerosis was to determine whether a relationship exists between the levels of plasma markers of leukocyte activation. i.e. cytokines and proteases and risk factors for ***atherosclerosis*** or the degree of atherosclerotic disease. Using ELISAs we measured the plasma levels of neutrophil gelatinase-associated lipocalin (NGAL), neutrophil protease 4 (NP4) as markers for neutrophil activation, tumor necrosis factor alpha (TNF) and soluble TNF receptor-1 (sTNFR-1) as markers of monocyte/ macrophage activation in 156 subjects with asymptomatic carotid artery plaque detected at ultrasound examination. Plasma TNF and sTNFR-1 levels were found to correlate with systolic blood pressure ($r = 0.32$, $P < 0.04$ and $r = 0.22$, $P < 0.05$, respectively), plasma NGAL level to correlate with diastolic blood pressure ($r = 0.22$; $P < 0.005$), the plasma levels of sTNFR-1 and NGAL to correlate with age ($r = 0.28$, $P < 0.001$ and $r = 0.20$, $P < 0.05$, respectively). As compared with non-smokers ($n = 112$), smokers ($n = 43$) had higher plasma levels of TNF (2.9 vs. 1.4 mu g/l; $P < 0.02$) and of NP4 (27.5 vs. 23.4 mu g/l; $P < 0.05$). The plasma NGAL level was higher in hypertensive women (it = 7) than in normotensive women ($n = 85$) (109 vs. 87 mu g/l; $P < 0.05$). We thus demonstrated that, in subjects with asymptomatic early atherosclerosis, the plasma levels of markers of systemic leukocyte activation were correlated with age and blood pressure, and were higher in smokers and ***hypertensives*** . These results support the hypothesized relationship between the level of systemic leukocyte activation and risk factors for atherosclerotic vascular disease. (C) 1997 Elsevier Science Ireland Ltd.

6/7/26 (Item 14 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05784955 Genuine Article#: WX244 Number of References: 119
Title: Review article: Platelets in inflammatory bowel disease - Pathogenetic role and therapeutic implications
Author(s): Collins CE (REPRINT) ; Rampton DS
Corporate Source: ST MARYS HOSP,DEPT MED/LONDON W2//ENGLAND/ (REPRINT); ST BARTHOLOMEWS & ROYAL LONDON SCH MED & DENT,GI SCI RES UNIT/LONDON//ENGLAND/
Journal: ALIMENTARY PHARMACOLOGY & THERAPEUTICS, 1997, V11, N2 (APR), P237-247
ISSN: 0269-2813 Publication date: 19970400
Publisher: BLACKWELL SCIENCE LTD, OSNEY MEAD, OXFORD, OXON, ENGLAND OX2 0EL
Language: English Document Type: REVIEW
Abstract: An elevated platelet count is well recognized as a marker of inflammatory bowel disease activity, There is an increased incidence of systemic thromboembolism in this disease. Recent work indicates that platelets exhibit several proinflammatory properties including release of inflammatory mediators, and recruitment, chemotaxis and modulation of the activity of other inflammatory cells. Furthermore there is evidence that microvascular thrombosis and a procoagulant state may play a role in the pathogenesis of inflammatory bowel disease.
These observations prompted recent studies of platelet activity in inflammatory bowel disease, which indicate enhanced platelet

aggregation in vivo and in vitro, and increased platelet activation as measured by increased release of intracellular proteins into plasma and expression of platelet surface markers, including P - ***selectin*** and GP53. These abnormalities could contribute to the pathogenesis of inflammatory bowel disease by enhancing inflammation and promoting microinfarction. Aminosalicylates reduce platelet activity although they also have many other additional properties to explain their efficacy in inflammatory bowel disease, There are however several specific antiplatelet drugs now available which may provide new therapeutic possibilities in the management of this disease.

6/7/27 (Item 15 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05624757 Genuine Article#: WL697 Number of References: 187
Title: Vascular endothelial dysfunction
Author(s): DeMeyer GRY (REPRINT) ; Herman AG
Corporate Source: UNIV INSTELLING ANTWERP,DIV PHARMACOL, UNIV PL 1/B-2610 WILRIJK//BELGIUM/ (REPRINT)
Journal: PROGRESS IN CARDIOVASCULAR DISEASES, 1997, V39, N4 (JAN-FEB) , P325-342
ISSN: 0033-0620 Publication date: 19970100
Publisher: W B SAUNDERS CO, INDEPENDENCE SQUARE WEST CURTIS CENTER, STE 300, PHILADELPHIA, PA 19106-3399
Language: English Document Type: REVIEW

6/7/28 (Item 16 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05574338 Genuine Article#: WH486 Number of References: 115
Title: Nitric oxide synthase: Role in the genesis of vascular disease
Author(s): Cooke JP (REPRINT) ; Dzau VJ
Corporate Source: STANFORD UNIV,DIV CARDIOVASC MED/STANFORD//CA/94305 (REPRINT)
Journal: ANNUAL REVIEW OF MEDICINE, 1997, V48, P489-509
ISSN: 0066-4219 Publication date: 19970000
Publisher: ANNUAL REVIEWS INC, 4139 EL CAMINO WAY, PO BOX 10139, PALO ALTO, CA 94303-0139
Language: English Document Type: REVIEW
Abstract: The product of nitric oxide (NO) synthase is the most potent endogenous vasodilator known. NO not only is a potent vasodilator, it also inhibits platelet adherence and aggregation, reduces adherence of leukocytes to the endothelium, and suppresses proliferation of vascular smooth muscle cells. A number of disorders are associated with reduced synthesis and/or increased degradation of vascular NO. These include hypercholesterolemia, diabetes mellitus, hypertension, and tobacco use. The endothelial dysfunction caused by these disorders contributes to the alterations in vascular function and structure observed in these conditions. A reduction in the activity of vascular NO likely plays a significant role in the development of ***atherosclerosis*** . Insights into the mechanisms by which NO production or activity is altered in these states will lead to new therapeutic strategies in the treatment of a number of vascular disorders, including hypertension, atherosclerosis, restenosis, and thrombosis.

6/7/29 (Item 17 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05484950 Genuine Article#: WB840 Number of References: 37
Title: OXIDIZED LOW-DENSITY LIPOPROTEINS AND MICROVASCULAR RESPONSES TO ISCHEMIA-REPERFUSION
Author(s): LIAO LX; HARRIS NR; GRANGER DN
Corporate Source: LOUISIANA STATE UNIV,MED CTR,DEPT PHYSIOL & BIOPHYS,1501 KINGS HIGHWAY,POB 33932/SHREVEPORT//LA/71130; LOUISIANA STATE UNIV,MED CTR,DEPT PHYSIOL & BIOPHYS/SHREVEPORT//LA/71130
Journal: AMERICAN JOURNAL OF PHYSIOLOGY-HEART AND CIRCULATORY PHYSIOLOGY, 1996, V40, N6 (DEC), PH2508-H2514
ISSN: 0363-6135
Language: ENGLISH Document Type: ARTICLE
Abstract: The objective of this study was to determine whether ischemia and reperfusion (I/R) and/or chronic arterial hypertension potentiates the leukocyte-endothelial cell adhesion (LECA) and microvascular dysfunction elicited by oxidized low-density lipoproteins (ox-LDL). Mast cell degranulation, leukocyte adherence adn emigration, and albumin leakage were monitored in postcapillary venules of rat mesentery. Intra-arterial infusion of copper-oxidized LDL (Cu-LDL), at a concentration that does not directly affect the microvasculature, significantly enhanced the I/R-induced recruitment of adherent and emigrated leukocytes but does not affect the increased albumin leakage and mast cell degranulation responses normally observed after I/R. Infusion of a higher concentration of Cu-LDL in nonischemic mesentery of either normotensive Wistar-Kyoto or spontaneously hypertensive rats elicited significant yet similar increases in LECA, mast cell degranulation, and albumin leakage. These findings indicate that 1) ox-LDL act synergistically with I/R to promote leukocyte recruitment in postcapillary venules but without an accompanying exacerbation of albumin leakage, and 2) ox-LDL do not elicit a more intense inflammatory response in the microvasculature of hypertensive versus normotensive animals.

6/7/30 (Item 18 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05358261 Genuine Article#: BG58X Number of References: 621
Title: DIFFERENTIATED PROPERTIES AND PROLIFERATION OF ARTERIAL SMOOTH-MUSCLE CELLS IN CULTURE
Author(s): THYBERG J
Corporate Source: KAROLINSKA INST,DEPT MOL & CELL BIOL/S-17177 STOCKHOLM//SWEDEN/
Journal: INTERNATIONAL REVIEW OF CYTOLOGY-A SURVEY OF CELL BIOLOGY, 1996, V169, P183-265
ISSN: 0074-7696
Language: ENGLISH Document Type: REVIEW

6/7/31 (Item 19 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05230098 Genuine Article#: VJ676 Number of References: 54
Title: EXACERBATION OF ATHEROSCLEROSIS BY HYPERTENSION - POTENTIAL MECHANISMS AND CLINICAL IMPLICATIONS
Author(s): CHOBANIAN AV; ALEXANDER RW

Corporate Source: BOSTON UNIV,SCH MED,80 E CONCORD ST/BOSTON//MA/02118; EMORY UNIV,SCH MED/ATLANTA//GA/00000
Journal: ARCHIVES OF INTERNAL MEDICINE, 1996, V156, N17 (SEP 23), P 1952-1956
ISSN: 0003-9926
Language: ENGLISH Document Type: REVIEW
Abstract: Recent experimental data suggest marked similarities between the effects of hypertension and hypercholesterolemia on the arterial intima. Both conditions also seem to exert proinflammatory effects on the artery, resulting in the recruitment of monocytes into the intima. These effects may be due to production of oxygen-free radicals, which in turn may stimulate genes involved in the recruitment of inflammatory cells into the arterial wall. Plaque rupture and acute myocardial infarction are related to local accumulation of inflammatory cells in vulnerable areas of the plaque. Recent clinical trials using cholesterol-lowering or antihypertensive therapies have shown a decrease in cardiovascular events that may have resulted from withdrawal of inflammatory effects on the arterial wall. Angiotensin-converting enzyme inhibitors decrease the rat of myocardial infarction in patients with overt congestive heart failure or left ventricular dysfunction. These drugs probably affect several mechanisms related to the inhibition of angiotensin production and the potentiation of bradykinin and resultant enhancement of nitric oxide and prostacyclin. The mechanisms could include reversing the proinflammatory effects of angiotensin and hypercholesterolemia on the arterial wall. Future therapeutic strategies of vascular protection in hypertension may include direct attacks on proinflammatory or pro-oxidant vascular mechanisms.

05172790 Genuine Article#: VE769 Number of References: 44
Title: INCREASE IN NUMBER OF WEIBEL-PALADE BODIES AND ENDOTHELIN-1 RELEASE FROM ENDOTHELIAL-CELLS IN THE CADMIUM-TREATED RAT THORACIC AORTA
Author(s): DOI Y; OZAKA T; FUKUSHIGE H; FURUKAWA H; YOSHIZUKA M; FUJIMOTO S
Corporate Source: UNIV OCCUPAT & ENVIRONM HLTH,SCH MED,DEPT ANAT/KITAKYUSHU/FUKUOKA 807/JAPAN/; UNIV OCCUPAT & ENVIRONM HLTH,SCH HLTH SCI,DEPT NURSING/KITAKYUSHU/FUKUOKA 807/JAPAN/; KURUME UNIV,SCH MED,DEPT ANAT/KURUME/FUKUOKA 830/JAPAN/
Journal: VIRCHOWS ARCHIV-AN INTERNATIONAL JOURNAL OF PATHOLOGY, 1996 , V428, N6 (AUG), P367-373
ISSN: 0945-6317
Language: ENGLISH Document Type: ARTICLE
Abstract: Male rats received daily intraperitoneal injections of cadmium sulphate (2.0 mg/kg) for 3 (Cd-3 group), 6 (Cd-6 group) and 8 days (Cd-8 group). The blood samples were prepared for endothelin (ET)-1 assay, and the thoracic aorta was investigated by both electron microscopy and immunoelectron microscopy using anti ET-1 sera. The plasma ET-1 concentrations of both Cd-6 and Cd-8 groups increased significantly in a cumulative dose-dependent manner. The cadmium-treated rat aorta showed an increase in the number of Weibel-Palade (WP) bodies in endothelial cells, and degranulation and exocytosis of WP bodies occurred exclusively in the Cd-8 group. Immunoreaction for ET-1 was localized preferentially in WP bodies of both cadmium-treated and control groups, and in the rough endoplasmic reticulum of the cadmium-treated groups only. Reactivity was also found on the WP bodies undergoing exocytosis in the Cd-8 group. Cadmium intoxication induces an increase in number of ET-1-storing WP bodies in

the rat aorta endothelium. The enhancement of extracellular release of their contents by exocytosis results in elevation of the plasma ET-1 concentration.

6/7/33 (Item 21 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05170303 Genuine Article#: VE928 Number of References: 59
Title: HYPERTENSION, THE ENDOTHELIUM AND THE PATHOGENESIS OF CHRONIC VASCULAR-DISEASE
Author(s): HALLER H
Corporate Source: HUMBOLDT UNIV BERLIN,VIRCHOW KLINIKUM,FRANZ VOLHARD KLIN,WILTBERG STR 50/D-13125 BERLIN//GERMANY/
Journal: KIDNEY & BLOOD PRESSURE RESEARCH, 1996, V19, N3-4, P166-171
ISSN: 1420-4096
Language: ENGLISH Document Type: ARTICLE
Abstract: The endothelium lines all vessels of the body and is the most important structure for communication between the flowing blood and the vessel wall. Healthy endothelium has antiadhesive and antithrombotic properties and is crucial for maintaining blood flow It serves as a permeability barrier and prevents noxious agents from entering the vessel wall. Endothelial cells have secretory functions and secrete vasorelaxant substances. Therefore, functioning endothelium sustains the homoeostasis of the vessel wall. Endothelial functions are impaired by risk factors for cardiovascular disease such as hypertension, hyperlipidemia and hyperglycemia. ***Hypertension*** leads to decreased generation of nitric oxide in endothelial cells, thereby diminishing their vasorelaxant properties. ***Hypertension*** also contributes to an increase in endothelial cell permeability leading to intimal edema. Thirdly, hypertension increases the expression of adhesion molecules and increases the adherence of leukocytes to the vessel wall. Hence, hypertension directly contributes to the pathological alterations of the endothelium and it seems that these effects initiate and accelerate the pathogenesis of chronic vascular disease.

6/7/34 (Item 22 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05042811 Genuine Article#: TL380 Number of References: 44
Title: DNA POLYMORPHISMS IN ADHESION MOLECULE GENES - A NEW RISK FACTOR FOR EARLY ATHEROSCLEROSIS
Author(s): WENZEL K; ERNST M; ROHDE K; BAUMANN G; SPEER A
Corporate Source: CHARITE,DEPT INTERNAL MED 1,DIV MOLEC BIOL,ZIEGELSTR 5-9/D-10117 BERLIN//GERMANY/; HUMBOLDT UNIV BERLIN,CHARITE,DEPT INTERNAL MED 1,DIV MOLEC BIOL/D-10098 BERLIN//GERMANY/; MAX DELBRUCK CTR MOLEC MED/D-13122 BERLIN//GERMANY/
Journal: HUMAN GENETICS, 1996, V97, N1 (JAN), P15-20
ISSN: 0340-6717
Language: ENGLISH Document Type: ARTICLE
Abstract: To contribute to the analysis of the genetic background of atherosclerosis, especially endothelial dysfunction, we searched for DNA polymorphisms in the genes encoding E-, P-, and L-selectin, and ICAM-I and VCAM-I. We detected 17 mutations by single-strand conformation polymorphisms analysis and direct sequencing. Five of them resulted in an amino acid substitution. In E-selectin, exchanges from serine to arginine (position 128), from leucine to phenylalanine (position 554), and a DNA mutation from guanine to thymine (position

98) present significantly different allele frequencies in young patients with angiographically established, severe ***atherosclerosis*** , compared with an unselected population. Results suggest that these polymorphisms are associated with a higher risk for early severe ***atherosclerosis*** .

6/7/35 (Item 23 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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04960932 Genuine Article#: UV164 Number of References: 34
Title: EVIDENCE FOR ACTIVATION OF ENDOTHELIUM AND MONOCYTES IN HYPERTENSIVE RATS
Author(s): LIU Y; LIU TN; MCCARRON RM; SPATZ M; FEUERSTEIN G; HALLENBECK JM ; SIREN AL
Corporate Source: UNIFORMED SERV UNIV HLTH SCI,DEPT NEUROL,4301 JONES BRIDGE RD/BETHESDA//MD/20814; UNIFORMED SERV UNIV HLTH SCI,DEPT NEUROL/BETHESDA//MD/20814
Journal: AMERICAN JOURNAL OF PHYSIOLOGY-HEART AND CIRCULATORY PHYSIOLOGY, 1996, V39, N6 (JUN), PH2125-H2131
ISSN: 0363-6135
Language: ENGLISH Document Type: ARTICLE
Abstract: We have proposed that an interaction between perivascular macrophages and endothelium via cytokines could underlie the increased risk of stroke in ***hypertension*** . Therefore, the activation of monocytes, the endothelial expression of intercellular adhesion molecule-1 (ICAM-1), and the numbers of monocytes/macrophages in carotid arteries, as well as the cytokine production in carotid tissue, of spontaneously hypertensive rats (SHR) and normotensive Wistar-Kyoto and Sprague-Dawley rats were studied. The total number of blood monocytes (890 +/- 153 cells/mm(3), n = 10) and the number of activated (nitro blue tetrazolium-positive monocytes (220 +/- 51 cells/mm(3), n = 10) were significantly greater ($P < 0.05$) in SHR than in WKY rats (440 +/- 81 and 40 +/- 16 cells/mm(3), respectively, n = 10). Patchy endothelial expression of ICAM-1 was found in 77 +/- 9% of carotid sections from stroke-prone SHR (SHR-SP, n = 5) and in 75 +/- 7% of the sections from SHR (n = 7) but in none of the sections from the two normotensive rat strains (n = 7). The number of endothelium-attached monocytes/macrophages per millimeter of internal elastic lamina was significantly greater in SHR-SP than in SHR [5.1 +/- 0.7 (n = 4) and 3.3 +/- 0.3 (n = 6), $P < 0.05$], whereas no monocytes were found around the endothelium in either of the normotensive rat strains (n = 7 in each group). Incubation of the carotid arteries with lipopolysaccharide (30-300 ng/ml) induced a concentration-dependent expression of mRNAs for interleukin-1 beta and release of tumor necrosis factor-alpha to a significantly greater degree in the SHR than in the Wistar-Kyoto rats. The results demonstrate that hypertension is associated with activation of monocytes and endothelium and an increased endothelial adhesion and subendothelial accumulation of monocytes/macrophages and with an increased vascular capacity to produce cytokines.

6/7/36 (Item 24 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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04805720 Genuine Article#: UJ126 Number of References: 25
Title: HYPERTENSION-ENHANCED MONOCYTE ADHESION IN EXPERIMENTAL ATHEROSCLEROSIS

Author(s): TROPEA BI; HUIE P; COOKE JP; TSAO PS; SIBLEY RK; ZARINS CK
Corporate Source: STANFORD UNIV HOSP,DIV VASC SURG,SUITE H-3600/STANFORD//CA/94305; STANFORD UNIV,SCH MED,DIV VASC SURG/STANFORD//CA/94305; STANFORD UNIV,SCH MED,DEPT PATHOL/STANFORD//CA/94305; STANFORD UNIV,SCH MED,DIV CARDIOVASC MED/STANFORD//CA/94305
Journal: JOURNAL OF VASCULAR SURGERY, 1996, V23, N4 (APR), P596-605
ISSN: 0741-5214
Language: ENGLISH Document Type: ARTICLE
Abstract: Purpose: Hypertension is a known clinical risk factor for ***atherosclerosis*** . In experimental ***atherosclerosis*** , monocyte adhesion to the endothelial surface is enhanced and is considered to be an important early stage in plaque formation. We tested the hypothesis that hypertension enhances monocyte adhesion in experimental ***atherosclerosis*** .

Methods: Twenty-two New Zealand White rabbits were fed an atherogenic diet for 3 weeks to induce plaque formation. Aortic coarctation was created in eight rabbits by wrapping a Dacron band around the midportion of the descending thoracic aorta (stenosis group), whereas six rabbits underwent banding without aortic constriction (no stenosis group). Eight rabbits served as nonoperated controls. Monocyte binding to the aortic endothelial surface was counted with epifluorescent microscopy on standard aortic segments proximal and distal to the band. Immunohistochemistry was performed for the following antibodies: VCAM-1, RAM11, CD11b, and factor VIII.

Results: Mean blood pressure was 89 +/- 3 mm Hg in the aorta proximal to the stenosis, compared with 64 +/- 4 mm Hg in the no stenosis group and 74 +/- 3 mm Hg in the control group ($P < 0.01$). The mean aortic blood pressure gradient across the stenosis was 16 +/- 2 mm Hg in the stenosis group, whereas the aortic blood pressure gradient was 0.2 +/- 0.6 mm Hg in the no stenosis group and -0.3 +/- 0.4 mm Hg in the control group ($p < 0.001$). Monocyte adhesion to the aortic endothelial surface proximal to the stenosis was increased twofold compared with adhesion to the aorta distal to the stenosis and compared with the proximal aorta in the control group ($P < 0.02$). The proximal-to-distal aortic ratio of monocyte binding was enhanced in the stenosis group (2.2) compared with the no stenosis (0.76) and control (0.83) groups ($p < 0.01$). The intima area of the aorta proximal to the stenosis was significantly increased compared with the proximal aortas in the no stenosis and control groups ($P < 0.01$). RAM11, CD11b, and endothelial VCAM-1 expression were enhanced in the hypertensive region proximal to the stenosis.

Conclusions: In the hypertensive region in the aorta proximal to the stenosis, monocyte adhesion and endothelial VCAM-1 expression were increased, with intimal thickening and accumulation of macrophages. These findings suggest that ***hypertension*** may promote atherosclerotic plaque formation by enhancing monocyte adhesion.

00822422 EMCare No: 30786493
Angiotensin II induces leukocyte-endothelial cell interactions in vivo via ATSUB1 and ATSUB2 receptor-mediated P-selectin upregulation
Piqueras L.; Kubes P.; Alvarez A.; O'Connor E.; Issekutz A.C.; Esplugues J.V.; Sanz M.-J.

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Circulation (CIRCULATION) (United States) 24 OCT 2000, 102/17 (2118-2123)
CODEN: CIRCA ISSN: 0009-7322
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 26
RECORD TYPE: Abstract

Background - Angiotensin II (Ang II) plays a critical role in the development of vascular lesions in hypertension, ***atherosclerosis*** , and several renal diseases. Because Ang II may contribute to the leukocyte recruitment associated with these pathological states, the aim of the present study was to assess the role of Ang II in leukocyte-endothelial cell interactions in vivo. Methods and Results - Intravital microscopy of the rat mesenteric postcapillary venules was used. Sixty minutes of superfusion with 1 nmol/L Ang II induced a significant increase in leukocyte rolling flux (83.8+/-20.7 versus 16.4+/-3.1 cells/min), adhesion (11.4+/-1.0 versus 0.8+/-0.5 cells/100 mum), and emigration (4.0+/-0.7 versus 0.2+/-0.2 cells/field) without any vasoconstrictor activity. These effects were not mediated by mast cell activation. Intravenous pretreatment with ATSUB1 (losartan) or ATSUB2 (PD123,319) receptor antagonists significantly reduced Ang II-induced responses. A combination of both receptor antagonists inhibited the leukocyte rolling flux, adhesion, and extravasation elicited by Ang II at 60 minutes. Pretreatment of animals with fucoidin or an adhesion-blocking anti-rat P-selectin monoclonal antibody abolished Ang II-induced leukocyte responses. Furthermore, rat platelet ***P*** - ***selectin*** expression was not affected by Ang II stimulation. Conclusions - Ang II induces significant leukocyte rolling, adhesion, and emigration, which may contribute not only to hypertension but also to the onset and progression of the vascular damage associated with disease states in which plasma levels of this peptide are elevated.

01583153 2000242816
Angiotensin II induces leukocyte-endothelial cell interactions in vivo via ATinf 1 and ATinf 2 receptor-mediated P-selectin upregulation
Piqueras L.; Kubes P.; Alvarez A.; O'Connor E.; Issekutz A.C.; Esplugues J.V.; Sanz M.-J.
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PUBLICATION DATE: October 24, 2000
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ISSN: 0009-7322
DOCUMENT TYPE: Article
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NO. OF REFERENCES: 26

Background - Angiotensin II (Ang II) plays a critical role in the development of vascular lesions in hypertension, ***atherosclerosis*** , and several renal diseases. Because Ang II may

contribute to the leukocyte recruitment associated with these pathological states, the aim of the present study was to assess the role of Ang II in leukocyte-endothelial cell interactions in vivo. Methods and Results - Intravital microscopy of the rat mesenteric postcapillary venules was used. Sixty minutes of superfusion with 1 nmol/L Ang II induced a significant increase in leukocyte rolling flux (83.8+/-20.7 versus 16.4+/-3.1 cells/min), adhesion (11.4+/-1.0 versus 0.8+/-0.5 cells/100 mum), and emigration (4.0+/-0.7 versus 0.2+/-0.2 cells/field) without any vasoconstrictor activity. These effects were not mediated by mast cell activation. Intravenous pretreatment with ATinf 1 (losartan) or ATinf 2 (PD123,319) receptor antagonists significantly reduced Ang II-induced responses. A combination of both receptor antagonists inhibited the leukocyte rolling flux, adhesion, and extravasation elicited by Ang II at 60 minutes. Pretreatment of animals with fucoidin or an adhesion-blocking anti-rat P-selectin monoclonal antibody abolished Ang II-induced leukocyte responses. Furthermore, rat platelet ***P*** - ***selectin*** expression was not affected by Ang II stimulation. Conclusions - Ang II induces significant leukocyte rolling, adhesion, and emigration, which may contribute not only to hypertension but also to the onset and progression of the vascular damage associated with disease states in which plasma levels of this peptide are elevated.

0078325048 EMBASE No: 2000374651

Angiotensin II induces leukocyte-endothelial cell interactions in vivo via AT SUB 1 and AT SUB 2 receptor-mediated P-selectin upregulation

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Circulation (Circulation) (United States) October 24, 2000, 102/17 (2118-2123)
CODEN: CIRCA ISSN: 0009-7322
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 26

Background - Angiotensin II (Ang II) plays a critical role in the development of vascular lesions in hypertension, ***atherosclerosis*** , and several renal diseases. Because Ang II may contribute to the leukocyte recruitment associated with these pathological states, the aim of the present study was to assess the role of Ang II in leukocyte-endothelial cell interactions in vivo. Methods and Results - Intravital microscopy of the rat mesenteric postcapillary venules was used. Sixty minutes of superfusion with 1 nmol/L Ang II induced a significant increase in leukocyte rolling flux (83.8+/-20.7 versus 16.4+/-3.1 cells/min), adhesion (11.4+/-1.0 versus 0.8+/-0.5 cells/100 mum), and emigration (4.0+/-0.7 versus 0.2+/-0.2 cells/field) without any vasoconstrictor activity. These effects were not mediated by mast cell activation. Intravenous pretreatment with AT SUB 1 (losartan) or AT SUB 2 (PD123,319) receptor antagonists significantly reduced Ang II-induced responses. A combination of both receptor antagonists inhibited the

leukocyte rolling flux, adhesion, and extravasation elicited by Ang II at 60 minutes. Pretreatment of animals with fucoidin or an adhesion-blocking anti-rat P-selectin monoclonal antibody abolished Ang II-induced leukocyte responses. Furthermore, rat platelet ***P*** - ***selectin*** expression was not affected by Ang II stimulation. Conclusions - Ang II induces significant leukocyte rolling, adhesion, and emigration, which may contribute not only to hypertension but also to the onset and progression of the vascular damage associated with disease states in which plasma levels of this peptide are elevated.

6/7/40 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

0077988152 EMBASE No: 2000037327
Role of transforming growth factor-beta1 in cardiovascular inflammatory changes induced by chronic inhibition of nitric oxide synthesis
Koyanagi M.; Egashira K.; Kubo-Inoue M.; Usui M.; Kitamoto S.; Tomita H.; Shimokawa H.; Takeshita A.
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Hypertension (Hypertension) (United States) January 1, 2000, 35/1 I (86-90)
CODEN: HPRTD ISSN: 0194-911X
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 29

We previously reported that chronic inhibition of nitric oxide (NO) synthesis with N(omega)-nitro-L-arginine methyl ester (L-NAME) induces inflammatory changes (monocyte infiltration, myofibroblast formation; and monocyte chemoattractant protein-1 [MCP-1] and transforming growth factor-beta [TGF-beta] expression) in the rat heart and vessel. There is debate regarding whether TGF-beta1 exhibits proinflammatory or anti-inflammatory activities. We used the rat model to investigate the role of TGF-beta in the pathogenesis of such inflammatory changes. We show here that infiltrating monocytes and myofibroblasts in the inflammatory lesions produced TGF-beta1 on the third day of L-NAME administration. Cotreatment with a monoclonal antibody against TGF- beta1, but not with control IgG, prevented the L-NAME-induced cardiac inflammation. The antibody also significantly inhibited the gene expression of MCP-1, P- ***selectin*** , and intercellular adhesion molecule-1. In summary, the antibody against TGF-beta1 prevented inflammatory changes in rat heart and vessel induced by chronic inhibition of NO synthesis, suggesting that increased production of TGF-beta1 is involved in the inflammatory changes in this model.

6/7/41 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE

0076878305 EMBASE No: 1997171390
Soluble P-selectin in hyperlipidaemia with and without

symptomatic vascular disease: Relationship with von Willebrand factor
Blann A.D.; Goode G.K.; Miller J.P.; McCollum C.N.
Thromb. Haemostasis Vasc. Biol. U., University Department of Medicine, City Hospital, Birmingham, United Kingdom; Thromb. Haemostasia Vasc. Biol. U., University Department of Medicine, City Hospital, Dudley Road, Birmingham B18 7QH, United Kingdom
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Blood Coagulation and Fibrinolysis (BLOOD COAGUL. FIBRINOLYSIS) (United Kingdom) June 24, 1997, 8/3 (200-204)
CODEN: BLFIE ISSN: 0957-5235
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 29

Soluble P-selectin (CD62P) may arise from platelets, the endothelium, or both, and raised levels are found in those with thrombotic disease and ***atherosclerosis*** . To determine whether these increased levels in atherosclerosis are related to hypercholesterolaemia, blood samples were obtained from 86 patients (43 with symptomatic vascular disease) attending a hypercholesterolaemia clinic, and 86 age- and sex-matched controls. Parallel measurement of endothelial cell product von Willebrand factor helped define the origin of sP-selectin. Using ELISAs, soluble P-selectin was higher (median 290 ng/ml, range 80-735, P < 005) in patients with vascular disease than in both patients with uncomplicated hypercholesterolaemia (median 210 ng/ml, range 55-550), and controls (median 190 ng/ml, range 48-500). Von Willebrand factor was raised in both patients with uncomplicated hypercholesterolaemia (115 +/- 26 IU/dl, P < 0.05) and patients with hypercholesterolaemia and vascular disease (129 +/- 32 IU/dl. P < 0.02) compared with controls (102 +/- 30 IU/dl). Levels of soluble ***P*** - ***selectin*** did not correlate with yon Willebrand factor, total low-density-lipoprotein (LDL) or high-density-lipoprotein (HDL) cholesterol or triglycerides levels, blood pressure or smoking, but von Willebrand factor correlated with LDL cholesterol (r = 042, P < 005). We conclude that plasma lipoproteins are not a major influence on levels of soluble ***P*** - ***selectin*** .

6/7/42 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13602692 PMID: 10833791
[Soluble P-selectin - a marker of platelet activation and vessel wall injury: increase of soluble P-selectin in plasma of patients with myocardial infarction, massive atherosclerosis and primary pulmonary hypertension]
Rastvorimui P-selektin - marker aktivatsii trombotsitov i porazheniia sosudistoi stenki: povyshenie ego urovnia v plazme krovi pri infarkte miokarda, rasprostranennom ateroskleroze i pervichnoi legochnoi gipertonii.
Semenov A V; Kogan-Ponomarev M Ia; Ruda M Ia; Komarov A L; Panchenko E P; Chazova I E; Mazurov A V
Terapevticheskii arkhiv (RUSSIA) 2000, 72 (4) p15-20, ISSN 0040-3660--Print Journal Code: 2984818R
Publishing Model Print
Document type: Comparative Study; English Abstract; Journal Article
Languages: RUSSIAN
Main Citation Owner: NLM
Record type: MEDLINE; Completed
AIM: A comparative analysis of the content of the soluble form of cell

adhesion protein P-selectin in the blood plasma of patients with acute myocardial infarction (AMI), massive atherosclerosis (MA) and primary pulmonary hypertension (PPH), investigation of the relationship between plasma content of P-selectin and known markers of platelets and endothelial cells activation, preliminary assessment of the prognostic value of ***P*** - ***selectin*** determination. MATERIALS AND METHODS: This study included 16 patients with AMI, 20 patients with MA, 21 patients with PPH and 18 healthy donors. The follow-up was 1-5 years. End-points in the group of patients with AMI were recurrent acute coronary syndrome and coronary artery by-pass operation, in the group with MA--thrombotic complications (acute coronary syndrome, ischemic stroke) and in the group with PPH--death. ***P*** - ***selectin*** was measured by ELISA and platelet factor 4 (PF4), thromboxane B2 (TXB2), endothelin-I and stable prostacyclin metabolite 6-keto-prostaglandin F1 alpha (6-keto-PGF1 alpha) by means of commercial ELISA kits. RESULTS: Mean level of P-selectin in blood plasma of patients with AMI (1 day) (361 +/- 18 ng/ml), MA (410 +/- 31 ng/ml) and PPH (627 +/- 83 ng/ml) was increased in comparison with the group of healthy donors (269 +/- 12 ng/ml) (everywhere p < 0.001). In AMI, ***P*** - ***selectin*** was increased on day 1 only, on days 2, 3 and 10-14 of the disease the level of P-selectin was significantly lower than on day 1 and did not differ from the control level in the group of donors. In patients with MA a significant correlation was detected between plasma content of P-***selectin*** and platelet activation marker PF4 (r = 0.606, P = 0.007) and in patients with PPH between the content of P-selectin and another platelet activation marker TXB2 (r = 0.622, p = 0.013). However, no correlation was found in PPH patients between the content of P-selectin and markers of endothelial activation and/or damage (endothelin-1 and 6-keto-PGF1 alpha). Difference in the concentration of P-selectin in patients with or without end-points during the follow-up period was detected in patients with AMI (353 +/- 14 ng/ml and 451 +/- 24 ng/ml, p = 0.009) and PPH (477 +/- 58 ng/ml and 927 +/- 184 ng/ml, p = 0.017) but not with MA (426 +/- 37 ng/ml and 361 +/- 24 ng/ml, p = 0.295). CONCLUSION: The level of ***P*** - ***selectin*** in plasma was increased in patients with acute thrombosis (AMI, 1 day) as well as in patients without clinical signs of thrombosis but with a massive injury of the vasculature (MA and PPH). The increase of ***P*** - ***selectin*** was, presumably, caused by its secretion from activated platelets since its concentration in plasma correlated with platelet concentration but not endothelial activation markers. Preliminary data indicate that blood plasma soluble P-selectin may be considered as a potential prognostic marker in AMI and PPH.
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Record Date Completed: 20000626

6/7/43 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12666370 PMID: 9588073
[Vascular endothelium as a factor in information transfer between the cardiovascular and immune systems]
Cievny endotel ako operator prenosu informacii medzi kardiovaskularnym a imunitnym systemom.
Stvrtinova V; Ferencik M; Hulin I; Jahnova E
II. interna klinika Lekarskej fakulty Univerzity Komenskeho v Bratislave.
Bratislavske lekarske listy (SLOVAKIA) Jan 1998, 99 (1) p5-19,
ISSN 0006-9248--Print Journal Code: 0065324
Publishing Model Print
Document type: English Abstract; Journal Article; Review

Languages: SLOVAK
Main Citation Owner: NLM
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In health, the vascular endothelium forms a multifunctional interface between the circulating blood and various tissues and organs of the body. It constitutes a selectively permeable barrier for macromolecules, as well as a nonthrombogenic and nonadhesive container that actively maintains the fluidity of blood. It is a metabolically active endocrine organ, serving as the source of multiple factors and mediators that are critical for normal homeostasis. These include vasodilators (nitric oxide, prostacyclin, endothelium-derived hyperpolarizing factor), vasoconstrictors (endothelin-1, thromboxane A2, prostaglandin H2 and components of the renin angiotensin system), various pro- and antithrombotic factors (e.g. tissue factor, platelet activating factor--PAF, von Willebrand factor), fibrinolytic activators and inhibitors (e.g. tissue plasminogen activator, plasminogen activator inhibitor-1), potent arachidonate metabolites (prostanoids), leukocyte adhesion molecules (e.g. E-selectin, ***P*** - selectin , intercellular adhesion molecule-1--ICAM-1, vascular cell adhesion molecule-1--VCAM-1), and multiple cytokines with activities of growth stimulators and inhibitors, transforming growth factors, proinflammatory and antiinflammatory mediators, tumour necrosis factors and chemotactic factors (chemokines). Besides these essential activities controlling the cardiovascular system, the endothelial cells represent an important part of the immune system as well. They have a pivotal role in the initiation and development of defensive and damaging inflammatory responses. Therefore endothelium can be considered as being the central equipment for the mutual exchange of life important information between the cardiovascular and immune systems. This in turn is leading to rapid advances in understanding the pathogenesis of some of the most serious and most common diseases, including inflammation, atherosclerosis and ***hypertension*** . (Tab. 7, Ref. 89.) (89 Refs.)
Record Date Created: 19980617
Record Date Completed: 19980617

6/7/44 (Item 1 from file: 370)
DIALOG(R)File 370:Science

00500312 (USE 9 FOR FULLTEXT)
Molecular Therapies for Vascular Diseases
Gibbons, Gary H.; Dzau, Victor J.
The authors are at the Falk Cardiovascular Research Center, Stanford University School of Medicine, Stanford, CA 94305-5246, USA.
Science Vol. 272 5262 pp. 689
Publication Date: 5-03-1996 (960503) Publication Year: 1996
Document Type: Journal ISSN: 0036-8075
Language: English
Section Heading: Articles
Word Count: 3885

Abstract: Vascular disease is the most common cause of death in the industrialized world. Although significant progress has been made in treating these disorders, more therapeutic agents must be developed that effectively prevent, arrest, or reverse this disease. Recent insights into the pathogenesis of vascular disease have opened up a new frontier of molecular therapies that target molecules as diverse as adhesion molecules and transcription factors. The biological rationale for these new therapies and their prospects for success are discussed.
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33. Supported by a Pew Biomedical Scholar award, the Baxter Foundation, the American Heart Association, and National Institutes of Health (NIH) grant HL-48638 (G.H.G.) and by NIH grants HL-35610, HL-35252, and HL-42663 (V.J.D.).

6/7/45 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

130033498 CA: 130(4)33498z PATENT
Methods for preventing progressive tissue necrosis, reperfusion injury, bacterial translocation and adult respiratory distress syndrome by using dehydroepiandrosterone derivatives
INVENTOR(AUTHOR): Araneo, Barbara A.; Daynes, Raymond A.; Orlinska, Urszula; Farrukh, Imad S.
LOCATION: USA
ASSIGNEE: University of Utah Research Foundation; Pharmadigm, Inc.
PATENT: PCT International ; WO 9855074 A2 DATE: 19981210
APPLICATION: WO 98US11141 (19980603) *US 870234 (19970605)
PAGES: 55 pp. CODEN: PIXXD2 LANGUAGE: English
PATENT CLASSIFICATIONS:
CLASS: A61K-000/A
DESIGNATED COUNTRIES: AU; CA; JP DESIGNATED REGIONAL: AT; BE; CH; CY; DE ; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE
SECTION:
CA202004 Mammalian Hormones
IDENTIFIERS: tissue necrosis prevention dehydroepiandrosterone derivs tissue injury, cell adhesion prevention dehydroepiandrosterone derivs tissue injury, reperfusion injury prevention dehydroepiandrosterone derivs, bacterial translocation prevention dehydroepiandrosterone derivs, adult respiratory distress syndrome prevention dehydroepiandrosterone derivs
DESCRIPTORS:
Burn...
chem. and thermal; use of dehydroepiandrosterone derivs. for preventing or reducing loss of tissue viability caused by adhesion of neutrophils to endothelial cells in patients with tissue injuries
Heart diseases...
hyperactive coronary circulation; use of dehydroepiandrosterone derivs. for preventing or reducing loss of tissue viability caused by adhesion

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    of neutrophils to endothelial cells in patients with tiss
Ischemia...
    method for preventing or reducing the effects of ischemia using
    dehydroepiandrosterone derivs.
Blood cells... Cell adhesion... Platelet adhesion... Platelet(blood)...
Vascular endothelium...
    methods for preventing or reducing adherence of blood cells and
    platelets to endothelial cells using dehydroepiandrosterone derivs.
P-selectin...
    methods for preventing or reducing adherence of blood cells and
    platelets to endothelial cells using dehydroepiandrosterone derivs. in
    relation to expression of P-selectin
Antihypertensives... Pulmonary hypertension...
    methods for preventing or reducing pulmonary hypertension using
    dehydroepiandrosterone derivs.
Adult respiratory distress syndrome... Bacterial infection... Necrosis...
Reperfusion injury...
    methods for preventing progressive tissue necrosis, reperfusion injury,
    bacterial translocation and adult respiratory distress syndrome by
    using dehydroepiandrosterone derivs.
Atherosclerosis... Hemorrhagic shock... Myocardial infarction... Neutrophil
adhesion... Surgery... Trauma...
    use of dehydroepiandrosterone derivs. for preventing or reducing loss
    of tissue viability caused by adhesion of neutrophils to endothelial
    cells in patients with tissue injuries
  CAS REGISTRY NUMBERS:
53-43-0D  derivs., methods for preventing progressive tissue necrosis,
    reperfusion injury, bacterial translocation and adult respiratory
    distress syndrome by using dehydroepiandrosterone derivs.
53-43-0  521-17-5  651-48-9  1093-91-0  1232-73-1  105119-96-8  methods for
    preventing progressive tissue necrosis, reperfusion injury, bacterial
    translocation and adult respiratory distress syndrome by using
    dehydroepiandrosterone derivs.
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PLEASE ENTER A COMMAND OR BE LOGGED OFF IN 5 MINUTES
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Set     Items   Description
S1       1535   (HYPERTENSI?) AND (PSGL? OR P(W)SELECTIN OR PADGEM OR GMP(-
            W)140 OR GMP140)
S2        341   (HYPERTENSI?) AND (PSGL? OR P(W)SELECTIN OR PADGEM OR GMP(-
            W)140 OR GMP140)(10N)(INHIBIT? OR ANTAGONI? OR SUPPRESS? OR T-
            REAT? OR THERAP?)
S3        210   RD S2  (unique items)
S4        384   S1 AND PY<2001
S5        216   RD S4  (unique items)
S6         45   S5 AND (ATHEROSCLEROSIS)
? ds

Set     Items   Description
S1       1535   (HYPERTENSI?) AND (PSGL? OR P(W)SELECTIN OR PADGEM OR GMP(-
            W)140 OR GMP140)
S2        341   (HYPERTENSI?) AND (PSGL? OR P(W)SELECTIN OR PADGEM OR GMP(-
            W)140 OR GMP140)(10N)(INHIBIT? OR ANTAGONI? OR SUPPRESS? OR T-
            REAT? OR THERAP?)
S3        210   RD S2  (unique items)
S4        384   S1 AND PY<2001
S5        216   RD S4  (unique items)
S6         45   S5 AND (ATHEROSCLEROSIS)
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Set     Items   Description
S1       1535   (HYPERTENSI?) AND (PSGL? OR P(W)SELECTIN OR PADGEM OR GMP(-
            W)140 OR GMP140)
S2        341   (HYPERTENSI?) AND (PSGL? OR P(W)SELECTIN OR PADGEM OR GMP(-
            W)140 OR GMP140)(10N)(INHIBIT? OR ANTAGONI? OR SUPPRESS? OR T-
            REAT? OR THERAP?)
S3        210   RD S2  (unique items)
S4        384   S1 AND PY<2001
S5        216   RD S4  (unique items)
S6         45   S5 AND (ATHEROSCLEROSIS)
? s s2 and py<2001
Processing
Processed  10 of  25 files ...
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>>>One or more prefixes are unsupported
>>>  or undefined in one or more files.
Processed  20 of  25 files ...
Processing
Completed processing all files
          341  S2
       106616338  PY<2001
      S7       76  S2 AND PY<2001
? rd s7
      S8       42  RD S7  (unique items)
? t s8/7/all
>>>Format 7 is not valid in file 143

 8/7/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2008 The Thomson Corporation. All rts. reserv.

15853147   BIOSIS NO.: 200100024986
Increased plasma P-selectin and decreased thrombomodulin in pulmonary
  arterial hypertension were improved by continuous prostacyclin
  therapy
AUTHOR: Sakamaki Fumio (Reprint); Kyotani Shingo; Nagaya Noritoshi; Sato
  Nagato; Oya Hideo; Satoh Toru; Nakanishi Norifumi
AUTHOR ADDRESS: Division of Cardiology and Pulmonary Circulation,
  Department of Medicine, National Cardiovascular Center, 5-7-1
  Fujishirodai, Suita-shi, Osaka, 565-8565, Japan**Japan
JOURNAL: Circulation  102 (22): p2720-2725 November 28, 2000 2000
MEDIUM: print
ISSN: 0009-7322
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background-Thrombosis in situ related to endothelial cell injury
  may contribute to the development of pulmonary   ***hypertension***   (PH).
  P-selectin, a leukocyte adhesion receptor present in endothelial cells
  and platelets, reflects endothelial injury and platelet activation, and
  thrombomodulin (TM), a receptor for thrombin and a major anticoagulant
  proteoglycan on the endothelial membrane, reflects the anticoagulant
  activity of the endothelium. Methods and Results-To assess abnormal
  coagulation due to endothelial injury in patients with PH, plasma levels
  of soluble P-selectin and TM were measured in 32 patients with primary PH
  (PPH), 25 with secondary pulmonary arterial hypertension (sPAH), 31
  with pulmonary venous hypertension (PVH), and 17 healthy subjects
  (Control). These measurements were repeated after continuous infusion of
  prostacyclin in 15 patients with PPH and 3 with sPAH. P-selectin levels
  in both the sPAH and PPH groups were significantly higher than those in
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the Control and PVH groups (P<0.05). Plasma TM level in the PPH group was significantly lower than those in the other groups (P<0.01). After prostacyclin therapy, the lower TM level was increased and the higher ***P*** - ***selectin*** level was decreased (P<0.05). Conclusions-Decreased TM and increased P-selectin in PPH and sPAH may reflect in situ thrombosis due to endothelial injury. Prostacyclin may act not only as a vasodilator but also as an agent that improves endothelial injury and altered hemostasis in pulmonary arterial injury.

8/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15731378 BIOSIS NO.: 200000449691
Interactions between blood cells and retinal endothelium in endotoxic sepsis
AUTHOR: Tsujikawa Akitaka; Kiryu Junichi (Reprint); Yamashiro Kenji; Nonaka Atsushi; Nishijima Kazuaki; Honda Yoshihito; Ogura Yuichiro
AUTHOR ADDRESS: Department of Ophthalmology and Visual Sciences, Kyoto University Graduate School of Medicine, Sakyo-ku, Kyoto, 606-8507, Japan **Japan
JOURNAL: Hypertension (Baltimore) 36 (2): p250-258 August, 2000 2000
MEDIUM: print
ISSN: 0194-911X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Platelets and leukocytes are thought to play a leading role in the pathogenesis of many inflammatory conditions. To recruit flowing blood cells to the inflammatory region, it would be necessary for them to interact with vascular endothelial cells. Recently, many reports have indicated the resistance of spontaneous hypertensive rats (SHR) to endotoxic sepsis. Their resistance might be derived from suppressed interaction between these blood cells and endothelial cells. Therefore, SHR and age-matched Wistar-Kyoto rats (WKY) were induced with endotoxic sepsis by intravenous injection of lipopolysaccharide (LPS). At 4, 12, 24, and 48 hours after induction, leukocyte-endothelial interactions in the retina were evaluated in vivo with acridine orange digital fluorography. Fluorescently labeled platelets were also injected to investigate platelet-endothelial interactions in the retina in endotoxic sepsis. Leukocyte rolling in SHR after LPS injection was significantly suppressed; the maximum number of rolling leukocytes was reduced by 80.1% at 12 hours after LPS injection in SHR compared with WKY. Subsequent leukocyte infiltration into the vitreous cavity was significantly inhibited in SHR. Furthermore, platelet-endothelial interactions in the retina were also suppressed in SHR treated with LPS. The maximum numbers of rolling and adherent platelets were reduced by 59.5% and 62.6%, respectively, in SHR compared with WKY. In both strains, leukocyte- and platelet-endothelial interactions were substantially inhibited by the blocking of ***P*** - ***selectin*** . These ***suppressed*** interactions could contribute to the reduction of leukocyte- and platelet-mediated tissue injury in endotoxic sepsis in SHR, resulting in their resistance to endotoxemia.

8/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

15159686 BIOSIS NO.: 199900419346
Platelet hyperactivity in hypertensive older patients is controlled by lowering blood pressure
AUTHOR: Riondino Silvia; Pignatelli Pasquale; Pulcinelli Fabio M; Lenti Luisa; Di Veroli Claudio; Marigliano Vincenzo; Gazzaniga Pier Paolo (Reprint)
AUTHOR ADDRESS: Dipartimento di Medicina Sperimentale e Patologia, Universia degli Studi di Roma "La Sapienza", Viale Regina Elena 324, 00161, Roma, Italy**Italy
JOURNAL: Journal of the American Geriatrics Society 47 (8): p943-947 Aug., 1999 1999
MEDIUM: print
ISSN: 0002-8614
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: OBJECTIVE: Patients with hypertension tend to have a high prevalence of atherothrombotic accidents. Platelet hyperactivity is frequently associated with ***hypertension*** . Because the vascular disease associated with hypertension evolves over the years, we investigated platelet activity parameters in a population of older hypertensive patients with no other risk factors for cardiovascular disease. PARTICIPANTS: We studied 34 older, nonsmoking patients (mean age 74 +- 5 years) with uncomplicated hypertension before and after the normalization of blood pressure (BP) was achieved with the angiotensin-converting enzyme inhibitor quinapril alone or in combination with the Ca2+ ***antagonist*** nifedipine. MEASUREMENTS: Platelet aggregation, P-selectin (CD62) expression on the platelet surface, serum levels of Interleukin-1beta (IL-1beta) and of Interleukin-6 (IL-6), as well as plasma levels of soluble P-selectin and Endothelin-1 (ET-1), were analyzed. RESULTS: All platelet hyperactivity parameters werereduced significantly with the normalization of BP at the end of antihypertensive drug treatment (systolic/diastolic: 186.2 +- 2.7/103.4 +- 1.1 mm Hg vs 135.0 +- 1.3/85.9 U; 1.9 mm Hg; P < 001). Those factors more strictly associated with endothelium injury, such as ET-1 and IL-6, did not show variations. A significant correlation (Spearman Rank test) was observed among all platelet function parameters and blood pressure values. CONCLUSIONS: This study demonstrated that even in a population of older hypertensive patients with no other risk factor for atherogenic disease, normalization of blood pressure induces a significant reduction of the parameters of enhanced platelet hyperactivity independent of the action exerted, at the platelet level, by the antihypertensive drugs.

8/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14785175 BIOSIS NO.: 199900044835
Inhaled nitric oxide does not affect adenosine 5'-diphosphate-dependent platelet activation infants with persistent pulmonary hypertension of the newborn
AUTHOR: Christou Helen (Reprint); Magnani Barbarajean; Morse David S; Allred Elizabeth N; Van Marter Linda J; Wessel David L; Kourembanas Stella
AUTHOR ADDRESS: Dep. Pediatrics, Div. Newborn Med. Dev. Newborn Biol., Children's Hosp., 300 Longwood Ave., Enders 9, Boston, MA 02115, USA**USA
JOURNAL: Pediatrics 102 (6): p1390-1393 Dec., 1998 ***1998***
MEDIUM: print

ISSN: 0031-4005
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Objective. To investigate the effect of inhaled nitric oxide (NO) treatment in newborns with persistent pulmonary hypertension on adenosine 5'-diphosphate (ADP)-dependent platelet activation. Methods. After parental informed consent, infants with persistent pulmonary hypertension of the newborn were randomly assigned to receive conventional treatment (control group) or treatment with 40 parts per million of inhaled NO. Platelet activation was measured at time of entry and 30 minutes later by surface expression of P-selectin in response to increasing concentrations of the agonist ADP (0, 2, 5, 10, and 20 muM) using fluorescence-activated flow cytometry. Results. We examined 11 infants in the inhaled NO group and 13 in the control group. P-selectin expression, quantified as mean fluorescence, was not significantly different in the two groups of patients at baseline. Median percent change from baseline fluorescence was assessed using the Wilcoxon matched-pairs signed-rank test. At 30 minutes after enrollment there were no statistically significant changes from baseline fluorescence in either group of patients and at all ADP concentrations. Conclusion. Thirty minutes of exposure to 40 ppm of inhaled NO does not inhibit ADP-dependent platelet activation as measured by surface expression of P-selectin in infants with persistent pulmonary
hypertension of the newborn.

12665822 BIOSIS NO.: 199598133655
Modification of leukocyte adhesion in spontaneously hypertensive rats by adrenal corticosteroids
AUTHOR: Suzuki Hidekazu; Zweifach Benjamin W; Forrest Michael J; Schmid-Schoenbein Gert W (Reprint)
AUTHOR ADDRESS: Inst. Biomed. Eng., Univ. Calif. San Diego, 9500 Gilman Dr., La Jolla, CA 92093-0412, USA**USA
JOURNAL: Journal of Leukocyte Biology 57 (1): p20-26 1995 1995
ISSN: 0741-5400
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Leukocyte adhesion is a key factor in the pathogenesis of organ injury following a variety of stimuli. In this study we have addressed the role of leukocyte adhesion in hypertensives as a risk factor for organ injury. In the spontaneously ***hypertensive*** rat (SHR), the number of circulating leukocytes and their level of activation are significantly increased compared with its normotensive control, the Wistar-Kyoto rat (WKY). We have demonstrated that elevated levels of glucocorticoid in SHR suppress P-selectin-mediated leukocyte-endothelial interaction in the microcirculation. It is possible that the disturbance in leukocyte-endothelial interactions may result in an elevated number of leukocytes in the circulation. The aim of the present study was to investigate the contribution of the adrenal glands to the disturbance in leukocyte behavior in SHR by subjecting the animals to bilateral adrenalectomy and investigating the effect of hydrocortisone. In addition, we have studied by immunohistochemistry the expression of the endothelial adhesion molecule, P-selectin, in response

to histamine in the mesenteric venules of normal and adrenalectomized SHR and WKY. The elevated blood pressure, above-normal leukocyte counts, and elevated number of activated neutrophils (nitroblue tetrazolium test) in SHR were blunted after adrenalectomy. The blunted histamine-induced leukocyte-endothelial interaction in the mesenteric venules of SHR was restored after adrenalectomy. Treatment with hydrocortisone significantly attenuated the elevated leukocyte adhesion in the adrenalectomized SHR as well as in WKY. The ***suppressed*** ***P*** - ***selectin*** expression in SHR mesentery was restored after adrenalectomy. In conclusion, the subnormal leukocyte-endothelial interaction in response to an inflammatory stimulation in SHR is abolished after adrenalectomy, suggesting a relationship between the altered leukocyte adhesiveness and the adrenal corticosteroids in ***hypertensives*** .

8/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12606428 BIOSIS NO.: 199598074261
Impaired-leukocyte-endothelial cell interaction in spontaneously hypertensive rats
AUTHOR: Suzuki Hidekazu; Schmid-Schoenbein Geert W; Suematsu Makoto; Delano Frank A; Forrest Michael J; Miyasaka Masayuki; Zweifach Benjami W
AUTHOR ADDRESS: Inst. Biomedical Eng., Univ. Calif. San Diego, 9500 Gilman Dr., La Jolla,, CA 92093-0412, USA**USA
JOURNAL: Hypertension (Dallas) 24 (6): p719-727 1994 1994
ISSN: 0194-911X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Hypertension is associated with a progressive organ injury whose etiology remains largely speculative. An increasing database shows that activated leukocytes, while affording an important immune protection, may be a contributing factor to several of the pathogenetic features of the ***hypertension*** syndrome. The purpose of this study was to determine the extent to which the glucocorticoid pathway may be involved in the atypical kinetics of leukocytes in spontaneously hypertensive rats (SHR) compared with normotensive Wistar-Kyoto (WKY) rats. The typical venular leukocyte adhesion induced by histamine application was significantly lower in SHR, and a comparison of normalized leukocyte rolling velocity (V-WBC/V-RBC) showed the values to be significantly higher in SHR relative to WKY controls. This abnormal trend in adherent leukocyte numbers and in V-WBC/V-RBC values could be counteracted when SHR were pretreated with RU 486, a synthetic glucocorticoid inhibitor, and restored to the levels observed in WKY rats. Anti- ***P*** - ***selectin*** monoclonal antibody (PB1.3) attenuated in SHR and WKY rats the increment of adherent leukocyte numbers as well as the decrement of V-WBC/V-RBC value that developed under combined histamine and RU 486 superfusion. Furthermore, an anti-intercellular adhesion molecule-1 monoclonal antibody (1A29) served to attenuate the increment of adherent leukocyte number induced by a combination of histamine and RU 486 superfusion in WKY rats and SHR. The results indicate that the deficient leukocyte-endothelial cell interaction in SHR can be circumvented by a glucocorticoid inhibitor.

8/7/7 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

08179037 Genuine Article#: 254UD Number of References: 30
Title: Effect of sulfatide on acute lung injury during endotoxemia in rats
Author(s): Squadrito F (REPRINT) ; Bagnato G; Altavilla D; Ferlito M; Campo GM; Squadrito G; Urna G; Sardella A; Arlotta M; Minutoli L; Quartarone C; Saitta A; Caputi AP
Corporate Source: UNIV MESSINA POLICLIN,INST PHARMACOL, SCH MED, TORRE BIOL 5 PIANO, VIA C VALERIA/I-98100 MESSINA//ITALY/ (REPRINT); UNIV MESSINA,DEPT INTERNAL MED, SCH MED/I-98100 MESSINA//ITALY/; UNIV MESSINA,SCH BIOL SCI, CHAIR PHARMACOL/I-98100 MESSINA//ITALY/
Journal: LIFE SCIENCES, 1999, V65, N24 (NOV 5), P2541-2552
ISSN: 0024-3205 Publication date: 19991105
Publisher: PERGAMON-ELSEVIER SCIENCE LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD OX5 1GB, ENGLAND
Language: English Document Type: ARTICLE
Abstract: Experimental studies have shown that intrapulmonary leukocyte sequestration and activation is implicated in the pathogenesis of acute lung injury during endotoxemia. Selectins are involved in the adhesion of leukocyte to the endothelium. Sulfatide is recognized by P selectin and blocks this adhesion molecule. We studied the effects of sulfatide on endotoxin-induced lung damage in rats. Endotoxin shock was produced in male rats by a single intravenous (i.v.) injection of 20 mg/kg of Salmonella enteritidis lipopolysaccharide (LPS). LPS administration reduced survival rate (0%, 72 h after endotoxin challenge) decreased mean arterial blood pressure (MAP), produced leukopenia (Controls=11,234 +/- 231 cells/mL, LPS=4,567 +/- 123 cells/mL) and increased lung myeloperoxidase activity (MPO; a marker of leukocyte accumulation) in the lung (Controls =0.35 +/- 0.1 U/g/tissue; LPS= 10 +/- 1.2 U/g/tissue). Furthermore LPS administration markedly impaired the concentration-response curves for acetylcholine and sodium nitroprusside in isolated polmonary arterial rings. There was also an increased staining for P-selectin in the pulmonary arteries. Sulfatide ***treatment*** (10 mg/kg, 30 min. after LPS challenge), significantly protected against LPS-induced lethality (90% survival rate and 70% survival rate 24 h and 72 h after LPS injection), reduced LPS induced hypothension, reverted leukopenia (8,895 +/- 234 cells/ml) and lowered lung MPO activity (1.7 +/- 0.9 U/g/tissue). Furthermore sulfatide restored to control values the LPS-induced impairment in arterial pulmonary vasorelaxation and reduced P-selectin immunostaining. Our data indicate that sulfatide attenuates LPS-induced lung injury and protects against endotoxin shock.

8/7/8 (Item 2 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

07762517 Genuine Article#: 205LU Number of References: 49
Title: Leukocyte-endothelial cell interactions in nitric oxide synthase-deficient mice
Author(s): Lefer DJ (REPRINT) ; Jones SP; Girod WG; Baines A; Grisham MB; Cockrell AS; Huang PL; Scalia R
Corporate Source: LOUISIANA STATE UNIV,MED CTR, DEPT MOL & CELLULAR PHYSIOL, 1501 KINGS HIGHWAY/SHREVEPORT//LA/71130 (REPRINT); MASSACHUSETTS GEN HOSP,CARDIOVASC RES CTR/BOSTON//MA/02129; THOMAS JEFFERSON UNIV,DEPT PHYSIOL/PHILADELPHIA//PA/19107
Journal: AMERICAN JOURNAL OF PHYSIOLOGY-HEART AND CIRCULATORY PHYSIOLOGY, 1999, V45, N6 (JUN), PH1943-H1950
ISSN: 0363-6135 Publication date: 19990600
Publisher: AMER PHYSIOLOGICAL SOC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814

Language: English Document Type: ARTICLE
Abstract: Nitric oxide (NO) is known to be an important endogenous modulator of leukocyte-endothelial cell interactions within the microcirculation. We examined leukocyte rolling and adhesion under baseline conditions and following thrombin (0.25 U/ml) superfusion in the mesentery of wildtype, inducible NOS (iNOS)-deficient (-/-), neuronal NOS (nNOS) -/-, and endothelial cell NOS (ecNOS) -/- mice to further our understanding of NO and leukocyte function. Baseline leukocyte rolling (cells/min) was significantly elevated in both the nNOS -/- (30.0 +/- 4.0) and ecNOS -/- mice (67.0 +/- 12.0) compared with wild-type mice (11.0 +/- 1.4). In addition, baseline leukocyte adherence (cells/100 mu m of vessel). was also significantly elevated in the nNOS -/- (5.2 +/- 1.0) and ecNOS -/- (13.0 +/- 1.3) compared with wild-type animals (1.3 +/- 0.5). Deficiency of iNOS had no effect on baseline leukocyte rolling or adhesion in the mesentery. Baseline surface expression of P-selectin was observed in 68.0 +/- 9.0% of intestinal venules in ecNOS -/- mice compared with 10.0 +/- 2.0% in wild-type mice. Additional studies demonstrated that administration of an anti-P-selectin monoclonal antibody (RB40.34) or the soluble ***P*** -selectin ligand, PSGL-1, completely inhibited the increased rolling and firm adhesion response in nNOS -/- and ecNOS -/- mice. Transmigration of neutrophils into the peritoneum following thioglycollate injection was also significantly augmented in nNOS -/- and ecNOS -/- mice. These studies clearly indicate the NO derived from both nNOS and ecNOS is critical in the regulation of leukocyte-endothelial cell interactions.

8/7/9 (Item 3 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

07276847 Genuine Article#: 144JR Number of References: 25
Title: Inhaled nitric oxide does not affect adenosine 5'-diphosphate-dependent platelet activation in infants with persistent pulmonary hypertension of the newborn
Author(s): Christou H; Magnani B; Morse DS; Allred EN; VanMarter LJ; Wessel DL; Kourembanas S (REPRINT)
Corporate Source: CHILDRENS HOSP,DEPT PEDIAT, DIV NEWBORN MED, 300 LONGWOOD AVE, ENDERS 9/BOSTON//MA/02115 (REPRINT); CHILDRENS HOSP,DEPT PEDIAT, DIV DEV & NEWBORN BIOL/BOSTON//MA/02115; CHILDRENS HOSP,DEPT PEDIAT, DIV NEWBORN MED/BOSTON//MA/02115; CHILDRENS HOSP,DEPT ANESTHESIA/BOSTON//MA/02115; CHILDRENS HOSP,DEPT LAB MED & PATHOL/BOSTON//MA/02115; CHILDRENS HOSP,DEPT CARDIOL/BOSTON//MA/02115; CHILDRENS HOSP,DEPT NEUROL/BOSTON//MA/02115; HARVARD UNIV,SCH MED, BRIGHAM & WOMENS HOSP, DEPT NEWBORN MED/BOSTON//MA/; HARVARD UNIV,SCH MED, BRIGHAM & WOMENS HOSP, DEPT ANESTHESIA/BOSTON//MA/; HARVARD UNIV,SCH PUBL HLTH, DEPT BIOSTAT/BOSTON//MA/02115
Journal: PEDIATRICS, 1998, V102, N6 (DEC), P1390-1393
ISSN: 0031-4005 Publication date: 19981200
Publisher: AMER ACAD PEDIATRICS, 141 NORTH-WEST POINT BLVD, ELK GROVE VILLAGE, IL 60007-1098
Language: English Document Type: ARTICLE
Abstract: Objective. To investigate the effect of inhaled nitric oxide (NO) treatment in newborns with persistent pulmonary hypertension on adenosine 5'-diphosphate (ADP)-dependent platelet activation.

Methods, After parental informed consent, infants with persistent pulmonary hypertension of the newborn were randomly assigned to receive conventional treatment (control group) or treatment with 40 parts per million of inhaled NO. platelet activation was measured at

time of entry and 30 minutes later by surface expression of P-selectin in response to increasing concentrations of the agonist ADP (0, 2, 5, 10, and 20 mu M) using fluorescence-activated flow cytometry.

Results. We examined 11 infants in the inhaled NO group and 13 in the control group. P-selectin expression, quantified as mean fluorescence, was not significantly different in the two groups of patients at baseline. Median percent change from baseline fluorescence was assessed using the Wilcoxon matched-pairs signed-rank test. At 30 minutes after enrollment there were no statistically significant changes from baseline fluorescence in either group of patients and at all ADP concentrations.

Conclusion. Thirty minutes of exposure to 40 ppm of inhaled NO does not inhibit ADP-dependent platelet activation as measured by surface expression of P-selectin in infants with persistent pulmonary ***hypertension*** of the newborn.

8/7/10 (Item 4 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

06654837 Genuine Article#: ZH470 Number of References: 48
Title: Inhaled nitric oxide inhibits human platelet aggregation, P-selectin expression, and fibrinogen binding in vitro and in vivo
Author(s): Gries A (REPRINT) ; Bode C; Peter K; Herr A; Bohrer H; Motsch J; Martin E
Corporate Source: UNIV HEIDELBERG,DEPT ANESTHESIOL, NEUENHEIMER FELD 110/D-69120 HEIDELBERG//GERMANY/ (REPRINT); UNIV HEIDELBERG,DEPT CARDIOL/D-69120 HEIDELBERG//GERMANY/
Journal: CIRCULATION, 1998, V97, N15 (APR 21), P1481-1487
ISSN: 0009-7322 Publication date: 19980421
Publisher: WILLIAMS & WILKINS, 351 WEST CAMDEN ST, BALTIMORE, MD 21201-2436
Language: English Document Type: ARTICLE
Abstract: Background-Recent data suggest that inhaled NO can inhibit platelet aggregation. This study investigates whether inhaled NO affects the expression level and avidity of platelet membrane receptors that mediate platelet adhesion and aggregation.

Methods and Results-In 30 healthy volunteers. platelet-rich plasma was incubated with an air/5% CO2 mixture containing 0, 100, 450, and 884 ppm inhaled NO. ADP-and collagen-induced platelet aggregation, the membrane expression of P-selectin, and the binding of fibrinogen to the platelet glycoprotein (GP) IIb/IIIa receptor were determined before (t(0)) and during the 240 minutes of incubation. In addition, eight patients suffering from severe adult respiratory distress syndrome (ARDS) were investigated before and 120 minutes after the beginning of administration of 10 ppm inhaled NO. In vitro, NO led to a dose-dependent inhibition of both ADP-induced (3+/-3% at 884 ppm versus 70+/-6% at 0 ppm after 240 minutes; P<.001) and collagen-induced (13+/-5% versus 62+/-5%; P<.01) platelet aggregation. Furthermore, P-selectin expression (36+/-7% of t(0) value: P<.01) and fibrinogen binding (33+/-11%; P<.01) were inhibited. In patients with ARDS, after two who did not respond to NO inhalation with an improvement in oxygenation had been excluded, an increase in plasma cGMP, prolongation of in vitro bleeding time, and inhibition of platelet aggregation and P-selectin expression were observed, and fibrinogen binding was also ***inhibited*** (19+/-7% versus 30+/-8%; P<.05).

Conclusions-NO-dependent inhibition of platelet aggregation may be caused by a decrease in fibrinogen binding to the platelet GP IIb/IIIa receptor.

8/7/11 (Item 5 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2008 The Thomson Corp. All rts. reserv.

06221361 Genuine Article#: YC877 Number of References: 69
Title: Endothelial cell injury in cardiovascular surgery: An overview
Author(s): Verrier ED (REPRINT) ; Boyle EM
Corporate Source: UNIV WASHINGTON,DEPT SURG, DIV CARDIOTHORAC SURG, 1059 PACIFIC AVE NE, BOX 356310/SEATTLE//WA/98195 (REPRINT)
Journal: ANNALS OF THORACIC SURGERY, 1997, V64, N4,S (OCT), PS2-S8
ISSN: 0003-4975 Publication date: 19971000
Publisher: ELSEVIER SCIENCE INC, 655 AVENUE OF THE AMERICAS, NEW YORK, NY 10010
Language: English Document Type: ARTICLE
Abstract: In the last decade the endothelium has been shown to play a major role in regulating membrane permeability, lipid transport, vasomotor tone, coagulation, inflammation, and vascular wall structure. These critical endothelial cell functions are extremely sensitive to injury in the form of hypoxia, exposure to cytokines, endotoxin, cholesterol, nicotine, surgical manipulation, or hemodynamic shear stress. In response to injury endothelial cells become activated, tipping the balance of endothelial-derived factors to disrupt barrier function, and enhance vasoconstriction, coagulation, leukocyte adhesion, and smooth muscle cell proliferation. Although these responses likely exist as protective mechanisms, if the stimuli are severe the responses may become excessive, resulting in damaged tissue, impaired organ function, and an abnormal fibroproliferative response. Recent discoveries in the field of vascular biology have led to an expanded understanding of many of the complications of cardiovascular operations. Because of the wide impact endothelial cell dysfunction has on patients with cardiovascular disease, issues pertaining to endothelial biology are in the forefront of research that will affect the current and future practice of cardiothoracic surgery. (C) 1996 by The Society of Thoracic Surgeons.

8/7/12 (Item 6 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2008 The Thomson Corp. All rts. reserv.

06119911 Genuine Article#: XW362 Number of References: 255
Title: Hypoxic-ischemic brain injury in the newborn - Cellular mechanisms and potential strategies for neuroprotection
Author(s): duPlessis AJ (REPRINT) ; Johnston MV
Corporate Source: CHILDRENS HOSP,DEPT NEUROL/BOSTON//MA/02115 (REPRINT); HARVARD UNIV,SCH MED, DEPT NEUROL/BOSTON//MA/02115; JOHNS HOPKINS UNIV,SCH MED, DEPT NEUROL/BALTIMORE//MD/21205; JOHNS HOPKINS UNIV,SCH MED, DEPT PEDIAT/BALTIMORE//MD/21205; KENNEDY KRIEGER INST,/BALTIMORE//MD/
Journal: CLINICS IN PERINATOLOGY, 1997, V24, N3 (SEP), P627-&
ISSN: 0095-5108 Publication date: 19970900
Publisher: W B SAUNDERS CO, INDEPENDENCE SQUARE WEST CURTIS CENTER, STE 300, PHILADELPHIA, PA 19106-3399
Language: English Document Type: REVIEW
Abstract: Recent advances have delineated many of the complex cellular mechanisms of cerebral hypoxic-ischemic injury. These developments have created opportunities for the design of rational '' mechanism-based ''

strategies that target specific injurious processes. A number of neuroprotective agents have entered adult clinical trial and practice. For the newborn infant, progress in this areas has been judiciously delayed by toxicity concerns. Central to those safety concerns is the close relationship between mechanisms of hypoxic-ischemic cellular injury and normal developmental processes. These cellular mechanisms and the dilemmas facing the advance of this field are discussed. The likelihood of an effective single '' magic bullet '' neuroprotective strategy emerging in the near future appears remote. Rather more likely is the development of '' cocktail '' therapies that seek to exploit synergistic antagonism at multiple levels in the complex concentration of cellular events mediating hypoxic-ischemic cellular injury. However, such combination therapies will require elucidation of the complex inter-relationships between mechanisms of cellular injury, brain development, and the combination of therapies envisioned.

8/7/13 (Item 7 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

06083981 Genuine Article#: XU236 Number of References: 98
Title: Role of nitric oxide in retinal cell death
Author(s): Roth S (REPRINT)
Corporate Source: UNIV CHICAGO,DEPT ANESTHESIA & CRIT CARE, 5841 S MARYLAND, BOX MC-4028/CHICAGO//IL/60637 (REPRINT)
Journal: CLINICAL NEUROSCIENCE, 1997, V4, N5, P216-223
ISSN: 1065-6766 Publication date: 19970000
Publisher: WILEY-LISS, DIV JOHN WILEY & SONS INC, 605 THIRD AVE, NEW YORK, NY 10158-0012
Language: English Document Type: ARTICLE
Abstract: Nitric oxide synthase (NOS), the enzyme that catalyzes the formation of nitric oxide from L-arginine, exists in three major isoforms, neuronal, endothelial, and immunologic. Neuronal and immunologic NOS has been detected in the retina. Neuronal NOS may be responsible for producing nitric oxide in photoreceptors and bipolar cells. Nitric oxide stimulates guanylate cyclase of photoreceptor rod cells and increases calcium-channel currents, which may be significant in the photoresponse. inducible nitric oxide synthase, found in Muller cells and in retinal pigment epithelium, may be involved in normal phagocytosis of the retinal outer segment, in infectious and ischemic processes, and in the pathogenesis of diabetic retinopathy. Nitric oxide contributes to basal tone in the retinal circulation. To date, findings are conflicting with respect to its role in retinal autoregulation. During glucose and oxygen deprivation, nitric oxide may increase blood flow and prevent platelet aggregation, but it may also mediate the toxic effects of excitatory amino acid release. Nonspecific inhibition of NOS appears to protect the retina from ischemic damage, suggesting an important role of nitric oxide in the pathogenesis of retinal ischemic injury, and possible therapeutic approaches in patients with retinal vascular occlusion. (C) 1997 Wiley-Liss, Inc.

8/7/14 (Item 8 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05967529 Genuine Article#: XL048 Number of References: 29
Title: A possible role of cytokines in the formation of peritoneal dissemination
Author(s): Yonemura Y (REPRINT) ; Endou Y; Nojima M; Kawamura T; Fujita H;

Kaji M; Ajisaka H; Bandou E; Sasaki T; Yamaguchi T; Harada S; Yamamoto H
Corporate Source: KANAZAWA UNIV,SCH MED, DEPT SURG 2, 13-1 TAKARA MACHI/KANAZAWA/ISHIKAWA 920/JAPAN/ (REPRINT); KANAZAWA UNIV,DEPT EXPT THERAPEUT, INST CANC/KANAZAWA/ISHIKAWA 920/JAPAN/; KANAZAWA UNIV,DEPT ELECTRON MICROSCOPY/KANAZAWA/ISHIKAWA 920/JAPAN/; KANAZAWA UNIV,DEPT BIOCHEM/KANAZAWA/ISHIKAWA 920/JAPAN/
Journal: INTERNATIONAL JOURNAL OF ONCOLOGY, 1997, V11, N2 (AUG), P 349-358
ISSN: 1019-6439 Publication date: 19970800
Publisher: INT JOURNAL ONCOLOGY, C/O PROFESSOR D A SPANDIDOS, EDITORIAL OFFICE, 1, S MERKOURI ST, ATHENS 116 35, GREECE
Language: English Document Type: ARTICLE
Abstract: The earliest event in the formation of peritoneal dissemination is considered through the process of the attachment of intraperitoneal free cancer cells to the submesothelial basement membrane, exposed after contraction of mesothelial cells. We studied the mechanisms of the contraction of mesothelial cells using a. highly metastatic sell line (MKN-45-P) to the peritoneum. Four hours after intraperitoneal inoculation of MKN-45-P, mouse mesothelial cells began to contract, and submesothelial basement membrane was widely exposed after 24 h. The same changes developed four hours after i.p. injection of IL-6, TNF-alpha and IL-8, and were most prominently observed in mice treated with IL-8. However, no significant changes were observed after treatment of HGF, EGF and TGF-beta. Furthermore, IL-1 alpha, IL-6, IL-8, TNF and EGF increased the number of intercellular gaps of a human mesothelial cell monolayer, which was incubated on Matrigel coated dishes. Normal mesothelial cells form a contiguous monolayer of closely apposed polygonal cells, each of which had prominent and peripheral bands of F-actin. After incubation with IL-1 alpha, IL-6, IL-8, TNF and EGF, peripheral actin bands became indistinct and the central stress fibers became numerous. However, no significant changes were found in mesothelial cells, which were treated with TGF-beta and HGF. In addition, the number of attached MKN-45-P cells on a mesothelial cell monolayer after treatment of IL-1 alpha (0.1-1 ng/ml), IL-8 (10-100 ng/ml), and TNF-alpha (100 ng/ml) was significantly larger than that of control and TGF-beta significantly reduced the number of attached cells. Concentration of IL-8 in the serum-free medium of MKN-45-P cells was high (3.4 ng/ml), but IL-1 alpha, IL-6, TNF-alpha, TGF-beta, EGF and HGF could not be detected. None of these cytokines were detected in the conditioning medium of human mesothelial cells. Based on these results, mesothelial cell contraction may be mediated by IL-1 alpha, IL-6, IL-8, TNF-alpha, and EGF, and these cytokines may be produced from cancer cells and/or intraperitoneal inflammatory cells. In contrast, TGF-beta have an inhibitory effect on the mesothelial cell contraction and attachment of cancer cells to a mesothelial monolayer. The attachment of free cancer cells on the peritoneum may be controlled with these cytokines.

8/7/15 (Item 9 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05810817 Genuine Article#: WZ024 Number of References: 161
Title: The influence of cardiopulmonary bypass on cytokines and cell-cell communication
Author(s): Hill GE (REPRINT) ; Whitten CW; Landers DF
Corporate Source: UNIV NEBRASKA,MED CTR, DEPT ANESTHESIOL, 600 S 42 ST, BOX 984455/OMAHA//NE/68198 (REPRINT); UNIV NEBRASKA,MED CTR, DEPT INTERNAL MED/OMAHA//NE/68198; UNIV TEXAS,SW MED CTR, DEPT ANESTHESIOL & PAIN

MANAGEMENT/DALLAS//TX/75235
Journal: JOURNAL OF CARDIOTHORACIC AND VASCULAR ANESTHESIA, 1997, V11
, N3 (MAY), P367-375
ISSN: 1053-0770 Publication date: 19970500
Publisher: W B SAUNDERS CO, INDEPENDENCE SQUARE WEST CURTIS CENTER, STE
300, PHILADELPHIA, PA 19106-3399
Language: English Document Type: REVIEW
Abstract: Cardiopulmonary bypass (CPB) is characterized by systemic endotoxemia immediately after its onset as well as the systemic release of proinflammatory cytokines, including tumor necrosis factor-alpha and the interleukins 1 and 6. Recent studies document that increased morbidity and mortality rates correlate with elevated systemic concentrations of these proinflammatory cytokines during adult and neonatal sepsis, following thoracoabdominal aortic aneurysm repair, as well as following CPB, These proinflammatory cytokines induce increased neutrophil and endothelial surface adhesive molecule expression, thereby promoting enhanced neutrophil-endothelial adherence. Increased neutrophil-endothelial adherence and subsequent neutrophil organ binding are thought to be a ''final common pathway'' of organ injury during clinical inflammatory conditions. Proinflammatory cytokines also increase cellular expression of inducible nitric oxide synthase, thus increasing cellular production of nitric oxide, a known inflammatory mediator. This review discusses recent evidence of the adverse effects of proinflammatory cytokine release during CPB and therapeutic modalities that can reduce the systemic concentrations of these mediators of inflammation. Copyright (C) 1997 by W.B. Saunders Company.

8/7/16 (Item 10 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05774463 Genuine Article#: WW930 Number of References: 45
Title: Pretreatment with inhaled nitric oxide inhibits neutrophil migration and oxidative activity resulting in attenuated sepsis-induced acute lung injury
Author(s): Bloomfield GL (REPRINT) ; Holloway S; Ridings PC; Fisher BJ; Blocher CR; Sholley M; Bunch T; Sugerman HJ; Fowler AA
Corporate Source: VIRGINIA COMMONWEALTH UNIV,MED COLL VIRGINIA, DEPT INTERNAL MED/RICHMOND//VA/23298 (REPRINT); VIRGINIA COMMONWEALTH UNIV,MED COLL VIRGINIA, DEPT SURG/RICHMOND//VA/23298; VIRGINIA COMMONWEALTH UNIV,MED COLL VIRGINIA, DEPT PEDIAT/RICHMOND//VA/23298; VIRGINIA COMMONWEALTH UNIV,MED COLL VIRGINIA, DEPT RESP CARE/RICHMOND//VA/23298; VIRGINIA COMMONWEALTH UNIV,MED COLL VIRGINIA, DEPT ANAT/RICHMOND//VA/23298
Journal: CRITICAL CARE MEDICINE, 1997, V25, N4 (APR), P584-593
ISSN: 0090-3493 Publication date: 19970400
Publisher: WILLIAMS & WILKINS, 351 WEST CAMDEN ST, BALTIMORE, MD 21201-2436
Language: English Document Type: ARTICLE
Abstract: Objective: To determine if, and by what mechanisms, inhaled nitric oxide attenuates acute lung injury in a porcine model of adult respiratory distress syndrome induced by Gram-negative sepsis.

Design: Nonrandomized, controlled study.

Setting: Laboratory at a university medical center.

Subjects: Thirty pathogen-free Yorkshire swine (15 to 20 kg),

Interventions: Four groups of swine were anesthetized, mechanically ventilated, and studied for 5 hrs. Both control-nitric oxide and septic-nitric oxide animals received inhaled nitric oxide at 20 parts

per million throughout the study, Control (n = 10) and control-nitric oxide (n = 5) animals received a 1-hr infusion of sterile saline, Sepsis was induced in septic (n = 10) and septic-nitric oxide (n = 5) animals with a 1-hr intravenous infusion of live Pseudomonas aeruginosa.

Measurements and Main Results: Untreated septic animals developed a progressive decrease in Pac, that was prevented in septic-nitric oxide animals (73 +/- 4 vs. 214 +/- 23 torr [9.7 +/- 0.5 vs. 28.5 +/- 3.1 kPa], respectively, at 5 hrs, p <.05), Untreated septic animals showed a significant increase in bronchoalveolar lavage protein and neutrophil count at 5 hrs, compared with the baseline value, indicating acute lung injury. Septic-nitric oxide animals showed no significant increase in these parameters. Peripheral blood neutraphils from untreated septic animals and septic-nitric oxide animals exhibited significant (p < .05) up-regulation of CD18 receptor expression and oxidant activity (10.5 +/- 0.9 and 5.0 +/- 0.9 nmol of superoxide anion/10(6) neutrophils/10 mins, respectively) compared with both control and control nitric oxide animals (3.0 +/- 0.6 and 2.6 +/- 0.2 nmol of superoxide anion/10(6) neutrophils/10 mins, respectively), Also, priming for the oxidant burst at 5 hrs was decreased by 50% in septic-nitric oxide animals compared with untreated septic animals, Both untreated septic and septic-nitric oxide animals showed a significant increase in pulmonary arterial pressure at 30 mins (47.5 +/- 2.4 and 51.0 +/- 3.0 mm Hg, respectively), followed by a progressive decrease (32.8 +/- 2.6 and 31.3 +/- 5.4 mm Hg, respectively, at 5 hrs). Both of these changes were significant (p < .05) compared with baseline values and compared with the control groups, There was no significant difference in pulmonary arterial pressure or systemic arterial pressure at any time between untreated septic and septic-nitric oxide animals.

Conclusions: These results demonstrate that inhaled nitric oxide attenuates alveolar-capillary membrane injury in this porcine model of Gram negative sepsis but does not adversely affect systemic hemodynamics, The data suggest that inhaled nitric oxide preserves alveolar-capillary membrane integrity by the following means: a) inhibiting transendothelial migration of activated, tightly adherent neutrophils; and b) possibly by attenuating the neutrophil oxidant burst.

05695289 Genuine Article#: WQ628 Number of References: 26
Title: Effect of oligosaccharides on rejection and reperfusion injury after lung transplantation
Author(s): Brandt M (REPRINT) ; Boeke K; Phillips ML; Steinhoff G; Haverich A
Corporate Source: HANNOVER MED SCH,DEPT CARDIOVASC & THORAC SURG, CARL NEWBERG STR 1/D-30625 HANNOVER//GERMANY/ (REPRINT); CHRISTIAN ALBRECHTS UNIV KIEL,DEPT CARDIOVASC SURG/KIEL//GERMANY/
Journal: JOURNAL OF HEART AND LUNG TRANSPLANTATION, 1997, V16, N3 (MAR), P352-359
ISSN: 1053-2498 Publication date: 19970300
Publisher: MOSBY-YEAR BOOK INC, 11830 WESTLINE INDUSTRIAL DR, ST LOUIS, MO 63146-3318
Language: English Document Type: ARTICLE
Abstract: Background. During rejection and reperfusion injury, infiltration of leukocytes into the lung allograft is regulated by adhesion

molecules, especially selectins. Sialyl-Lewis X (SLX), an oligosaccharide, is a membrane ligand molecule of P-selectin adhesion receptors. In this study, we investigated the effect of intravenous administration of a synthetic oligosaccharide analog of SLX on rejection and reperfusion injury after rat lung transplantation.

Methods. Left lateral, orthotopic, allogeneic lung transplantation was performed between fully incompatible rat strains (Dark Agouti --> Lewis) after an average total ischemic time of 45 minutes. Group A (n = 6) served as control; no immunosuppression was used. In group B (n = 6), rats received 200 mu g/kg/day SLX intravenously on days 0 to 4. The animals were killed on days 5 and 10, respectively. In groups C and D, syngeneic lung transplantation was performed (Lewis --> Lewis), with an ischemic time of 7 hours. Group C (n = 6) served as untreated controls. Group D rats (n = 6) received a single dose of 20 mg/kg SLX at the end of the ischemic time. The animals were killed on days 2 and 5, respectively.

Results. In group B rats, treated for rejection, a lower grade of rejection (2.7 +/- 0.6 vs 4.0 +/- 0.0, p < 0.05) and fewer infiltrating CD11a-positive leukocytes (6.6 +/- 2.7 vs 18.6 +/- 7.3, p < 0.05) were found histologically compared with group A. In group D rats, treated for reperfusion injury, a significant reduction of reperfusion injury was detected on chest radiograms and by histologic study.

Conclusions. A synthetic oligosaccharide analog of SLX reduces allograft rejection and reperfusion injury by abrogation of P-***selectin*** -dependent leukocyte-endothelial interaction. According to these findings, treatment with oligosaccharides to reduce reperfusion injury and rejection seems to be a promising strategy for clinical lung transplantation.

8/7/18 (Item 12 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05657421 Genuine Article#: WN744 Number of References: 317
Title: Cytokines, adhesion molecules, antiendothelial cell autoantibodies and vascular disease
Author(s): Carvalho D; Savage C (REPRINT)
Corporate Source: UNIV BIRMINGHAM,SCH MED, RENAL IMMUNOBIOL GRP, CCRIS/BIRMINGHAM B15 2TT/W MIDLANDS/ENGLAND/ (REPRINT); UNIV BIRMINGHAM,SCH MED, RENAL IMMUNOBIOL GRP, CCRIS/BIRMINGHAM B15 2TT/W MIDLANDS/ENGLAND/; HOSP CURRY CABRAL,DEPT NEPHROL/LISBON//PORTUGAL/
Journal: CARDIOVASCULAR PATHOLOGY, 1997, V6, N2 (MAR-APR), P61-78
ISSN: 1054-8807 Publication date: 19970300
Publisher: ELSEVIER SCIENCE INC, 655 AVENUE OF THE AMERICAS, NEW YORK, NY 10010
Language: English Document Type: REVIEW
Abstract: Vascular endothelial cells present a surface to circulating blood elements that is continuously changing its phenotype under the influence of cytokines and other soluble mediators, and as a result of cell-cell interactions. Phenotypic changes enable endothelial cells to respond to local environmental conditions, for example, promoting pro-inflammatory or pro-coagulant properties. Antibodies may also develop to structures on this changing endothelial surface. The characteristics of antiendothelial cell antibodies identified to date and their perceived role in vascular disease is discussed. (C) 1997 by Elsevier Science Inc.

8/7/19 (Item 13 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05627259 Genuine Article#: WL824 Number of References: 99
Title: Mechanisms of tissue repair: From wound healing to fibrosis
Author(s): Mutsaers SE (REPRINT) ; Bishop JE; McGrouther G; Laurent GJ
Corporate Source: UNIV COLL LONDON,SCH MED, RAYNE INST, CTR CARDIOPULM BIOCHEM & RESP MED/LONDON//ENGLAND/ (REPRINT); UNIV COLL LONDON,SCH MED, RAYNE INST, DIV PLAST & RECONSTRUCT SURG/LONDON//ENGLAND/
Journal: INTERNATIONAL JOURNAL OF BIOCHEMISTRY & CELL BIOLOGY, 1997 , V29, N1 (JAN), P5-17
ISSN: 1357-2725 Publication date: 19970100
Publisher: PERGAMON-ELSEVIER SCIENCE LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD, ENGLAND OX5 1GB
Language: English Document Type: REVIEW
Abstract: To set the scene for this Directed Issue on Mechanisms of Tissue Repair of The international Journal of Biochemistry aad Cell Biology, this introductory overview briefly describes the process of wound healing and highlights some of the key recent advances in this field of research. It emphasizes the importance of cell-cell and cell-matrix interactions, particularly relating to the role of cell surface adhesion molecules, and describes developments that have led to a better understanding of the dynamic nature of matrix turnover with reference to negative and positive mediators that regulate procollagen gene expression and protein production. An important component of this Directed Issue is concerned with the development of tissue fibrosis, which accompanies a number of disease states and demonstrates remarkable parallels with the normal wound healing process; excessive amounts of matrix are laid down but the resolution of scarring, which would be anticipated in wound healing, is impaired. The possible mechanisms involved in fibrosis are discussed here. Since cytokines play an important role in regulating cell function such as proliferation, migration and matrix synthesis, it is the balance of these mediators which is likely to play a key role in regulating the initiation, progression and resolution of wounds. Finally, this review highlights areas of tissue repair research in which recent developments have important clinical implications that may lead to novel therapeutic strategies. (C) 1997 Published by Elsevier Science Ltd.

8/7/20 (Item 14 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05604223 Genuine Article#: WK408 Number of References: 51
Title: Peroxynitrite inhibits leukocyte-endothelial cell interactions and protects against ischemia-reperfusion injury in rats
Author(s): Lefer DJ; Scalia R; Campbell B; Nossuli T; Hayward R; Salamon M; Grayson J; Lefer AM (REPRINT)
Corporate Source: THOMAS JEFFERSON UNIV,JEFFERSON MED COLL, DEPT PHYSIOL, 1020 LOCUST ST/PHILADELPHIA//PA/19107 (REPRINT); THOMAS JEFFERSON UNIV,JEFFERSON MED COLL, DEPT PHYSIOL/PHILADELPHIA//PA/19107; TULANE UNIV,SCH MED, DEPT MED, CARDIOL SECT/NEW ORLEANS//LA/70112
Journal: JOURNAL OF CLINICAL INVESTIGATION, 1997, V99, N4 (FEB 15), P 684-691
ISSN: 0021-9738 Publication date: 19970215
Publisher: ROCKEFELLER UNIV PRESS, 1114 FIRST AVE, 4TH FL, NEW YORK, NY 10021
Language: English Document Type: ARTICLE
Abstract: Peroxynitrite (ONOO-) anion, formed by the interaction of

superoxide with nitric oxide (NO), has previously been implicated as a cytotoxic agent. However, the effects of this free radical species on neutrophil (PMN)-endothelial cell interactions is largely unknown. We investigated the direct actions of ONOO- on PMN adhesion to endothelial cells in vitro and in vivo, as well as the effects of ONOO- on PMN-mediated myocardial ischemia-reperfusion injury. In vitro, peroxynitrite (100-1,000 nM) inhibited the adhesion of rat PMNs to the endothelium of isolated thrombin- or H2O2-stimulated rat mesenteric artery (P < 0.01 vs. thrombin or H2O2 alone). In vivo, in the rat mesentery, thrombin (0.5 U/ml) or N-G-nitro-L-arginine-methyl ester (50 mu M) significantly increased venular leukocyte rolling and adherence, which were also significantly (P < 0.01) attenuated by ONOO- (800 nM) accompanied by reduced P-selectin expression on the endothelial cell surface. Isolated perfused rat hearts were subjected to global ischemia and reperfusion with rat PMNs (10(8) cells), which resulted in profound cardiac depression (i.e., a marked reduction in left ventricular developed pressure and maximal rate of development of left ventricular pressure). Infusion of ONOO- reversed the myocardial contractile dysfunction of ischemic-reperfused rat hearts to near baseline levels, and markedly attenuated the accumulation of PMNs in the postischemic heart. The present study provides strong evidence that nanomolar concentrations of ONOO- both inhibit leukocyte-endothelial cell interactions and exert cytoprotective effects in myocardial ischemia-reperfusion injury. Furthermore, our results suggest that the inhibition of P-selectin expression by peroxynitrite is a key mechanism of the modulatory actions of ONOO- on leukocyte-endothelial cell interactions.

8/7/21 (Item 15 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05598184 Genuine Article#: WJ556 Number of References: 164
Title: Cell adhesion molecules and asthma
Author(s): Bloemen PGM; Hendricks PAJ (REPRINT) ; Nijkamp FP
Corporate Source: UTRECHT INST PHARMACEUT SCI,DEPT PHARMACOL & PATHOPHYSIOL, POB 80082/NL-3508 TB UTRECHT//NETHERLANDS/ (REPRINT); UTRECHT INST PHARMACEUT SCI,DEPT PHARMACOL & PATHOPHYSIOL/NL-3508 TB UTRECHT//NETHERLANDS/
Journal: CLINICAL AND EXPERIMENTAL ALLERGY, 1997, V27, N2 (FEB), P 128-141
ISSN: 0954-7894 Publication date: 19970200
Publisher: BLACKWELL SCIENCE LTD, OSNEY MEAD, OXFORD, OXON, ENGLAND OX2 0EL
Language: English Document Type: REVIEW

8/7/22 (Item 16 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05319611 Genuine Article#: VQ180 Number of References: 48
Title: EFFECT OF MATRIX GLYCATION ON EXPRESSION OF TYPE-IV COLLAGEN, MMP-2, MMP-9 AND TIMP-1 BY HUMAN MESANGIAL CELLS
Author(s): ANDERSON SS; WU KJ; NAGASE H; STETTLERSTEVENSON WG; KIM YK; TSILIBARY EC
Corporate Source: UNIV MINNESOTA,SCH MED,DEPT LAB MED & PATHOL,BOX 609 UMHC,420 DELAWARE ST SE/MINNEAPOLIS//MN/55455; UNIV KANSAS,SCH MED,DEPT BIOCHEM & MOL BIOL/KANSAS CITY//KS/66160; NCI,PATHOL LAB,NIH/BETHESDA//MD/20892; UNIV MINNESOTA,SCH MED,DEPT PEDIAT/MINNEAPOLIS//MN/55455

Journal: CELL ADHESION AND COMMUNICATION, 1996, V4, N2, P89-101
ISSN: 1061-5385
Language: ENGLISH Document Type: ARTICLE
Abstract: Human mesangial cells grown in either 5 or 25 mM glucose were cultured on type IV collagen which had been previously control-incubated or in vitro glycated. Northern blot analysis revealed that after 3-7 days in culture mesangial cells on glycated type IV collagen expressed similar to 25-200% more alpha 1(IV), similar to 20-50%, less matrix metalloproteinase 2 (MMP-2), and 65-75% more tissue inhibitor of metalloproteinase 1 (TIMP-1). Decreased immunoreactivity (similar to 30-40%) and collagenolytic activity (similar to 10-40%) corresponding to MMP-2 was also detected in media conditioned during the third day of culture on glycated type IV collagen, These effects on cell function were related to the extent of type IV collagen modification and were similar for cells cultured in 5 or 25mM glucose. Elevated glucose (25 vrs 5mM) increased expression of alpha 1(IV) mRNA (similar to 40-70%) and in conjunction with matrix glycation resulted in detectable levels of MMP-9 message by northern blot although collagenolytic activity corresponding to MMP-9 was not detectable by zymography. We conclude that glucose and matrix glycation may each alter mesangial cell function, perhaps leading to an imbalance in mesangial matrix synthesis and degradation which could contribute to mesangial expansion characteristic of diabetic renal disease.

8/7/23 (Item 17 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05230098 Genuine Article#: VJ676 Number of References: 54
Title: EXACERBATION OF ATHEROSCLEROSIS BY HYPERTENSION - POTENTIAL MECHANISMS AND CLINICAL IMPLICATIONS
Author(s): CHOBANIAN AV; ALEXANDER RW
Corporate Source: BOSTON UNIV,SCH MED,80 E CONCORD ST/BOSTON//MA/02118; EMORY UNIV,SCH MED/ATLANTA//GA/00000
Journal: ARCHIVES OF INTERNAL MEDICINE, 1996, V156, N17 (SEP 23), P 1952-1956
ISSN: 0003-9926
Language: ENGLISH Document Type: REVIEW
Abstract: Recent experimental data suggest marked similarities between the effects of hypertension and hypercholesterolemia on the arterial intima. Both conditions also seem to exert proinflammatory effects on the artery, resulting in the recruitment of monocytes into the intima. These effects may be due to production of oxygen-free radicals, which in turn may stimulate genes involved in the recruitment of inflammatory cells into the arterial wall. Plaque rupture and acute myocardial infarction are related to local accumulation of inflammatory cells in vulnerable areas of the plaque. Recent clinical trials using cholesterol-lowering or antihypertensive therapies have shown a decrease in cardiovascular events that may have resulted from withdrawal of inflammatory effects on the arterial wall. Angiotensin-converting enzyme inhibitors decrease the rat of myocardial infarction in patients with overt congestive heart failure or left ventricular dysfunction. These drugs probably affect several mechanisms related to the inhibition of angiotensin production and the potentiation of bradykinin and resultant enhancement of nitric oxide and prostacyclin. The mechanisms could include reversing the proinflammatory effects of angiotensin and hypercholesterolemia on the arterial wall. Future therapeutic strategies of vascular protection in hypertension may include direct attacks on proinflammatory or pro-oxidant vascular mechanisms.

8/7/24 (Item 18 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05068745 Genuine Article#: TN398 Number of References: 183
Title: PATHOGENESIS AND TREATMENT OF THE ADULT-RESPIRATORY-DISTRESS-SYNDROME
Author(s): FULKERSON WJ; MACINTYRE N; STAMLER J; CRAPO JD
Corporate Source: DUKE UNIV,MED CTR,DIV PULM & CRIT CARE MED/DURHAM//NC/27706
Journal: ARCHIVES OF INTERNAL MEDICINE, 1996, V156, N1 (JAN 8), P 29-38
ISSN: 0003-9926
Language: ENGLISH Document Type: REVIEW
Abstract: The adult respiratory distress syndrome is an acute clinical illness characterized by noncardiogenic pulmonary edema and refractory hypoxemia. Injury to the alveolar-capillary barrier and lung inflammation lead to intrapulmonary shunting of blood, surfactant depletion, and pulmonary vascular obstruction, Numerous mediators contribute to the pathologic response, Conventional therapy includes treating underlying causes and positive pressure mechanical ventilation. Concern about pressure-induced lung injury had led to new strategies to accomplish adequate gas exchange. Novel therapeutic interventions have included extracorporeal support techniques, use of compounds designed to neutralize proinflammatory cytokines, and administration of surfactants, but these efforts have not definitely affected mortality in randomized trials. Potent antioxidant agents have shown promise in animal models of acute lung injury, but human studies are lacking. Inhaled nitric oxide appears to have temporary effects on pulmonary artery pressure and on ventilation or perfusion relationships, but longer-term efficacy and safety in patients suffering from adult respiratory distress syndrome is unknown and awaits results of ongoing clinical trials.

8/7/25 (Item 19 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

04834909 Genuine Article#: UL073 Number of References: 45
Title: CARDIOVASCULAR MYSTERY SERIES - STRETCHING THE EVIDENCE IN THE CASE OF CARDIAC GROWTH
Author(s): YAMAZAKI T; KOMURO I; NAGAI R; YAZAKI Y
Corporate Source: UNIV TOKYO,SCH MED,DEPT MED 3/TOKYO 113//JAPAN/; UNIV TOKYO,SCH MED,DEPT MED 3/TOKYO 113//JAPAN/
Journal: CARDIOVASCULAR RESEARCH, 1996, V31, N4 (APR), P493-498
ISSN: 0008-6363
Language: ENGLISH Document Type: REVIEW

8/7/26 (Item 20 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

04748409 Genuine Article#: TY334 Number of References: 86
Title: THE ROLE OF THROMBOSIS IN SEVERE PULMONARY-HYPERTENSION
Author(s): CHAOUAT A; WEITZENBLUM E; HIGENBOTTAM T
Corporate Source: HOP HAUTE PIERRE,SERV PNEUMOL,AVE MOLIERE/F-67098 STRASBOURG//FRANCE/; PAPWORTH HOSP,DEPT RESP

PHYSIOL/CAMBRIDGE//ENGLAND/; HOP HAUTE PIERRE,DEPT PULMONOL/STRASBOURG//FRANCE/
Journal: EUROPEAN RESPIRATORY JOURNAL, 1996, V9, N2 (FEB), P356-363
ISSN: 0903-1936
Language: ENGLISH Document Type: ARTICLE
Abstract: Considering the important surface in pulmonary circulation where blood can interact with the endothelium, the maintenance of blood fluidity through the lung, by antithrombotic pathways and products of the endothelium, is essential. This function appears to be ineffective in primary pulmonary hypertension and in severe secondary pulmonary ***hypertension*** . Thrombotic lesions are frequently found in pulmonary arteries in these diseases.

Thrombin activity appears to be increased in severe pulmonary ***hypertension*** . Antithrombotic pathway disorders may account for this abnormality, particularly in chronic thromboembolic pulmonary ***hypertension*** and primary pulmonary ***hypertension*** . Injured endothelium, a constant feature in severe pulmonary hypertension, either primary or secondary, enhances thrombus formation in pulmonary vessels. This is probably related to thrombomodulin and tissue factor imbalance, impairment of prostacyclin and nitric oxide release, as well as inefficiency of fibrinolysis. Moreover, platelets appear to be activated in the pulmonary circulation of these patients. They release several mediators acting on vascular tone and as mitogenic agents, and may also contribute to thrombin and dot generation. Long-term oral anticoagulant and continuous infusion of prostacyclin, treatments which impede thrombosis, are known to improve the survival rate in patients with primary pulmonary ***hypertension*** .

These are the strongest arguments, so far, in favour of the role of thrombosis in severe pulmonary ***hypertension*** . However, we do not know whether these abnormalities result from a previous vascular injury or represent the primary disturbance.

8/7/27 (Item 21 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

04252657 Genuine Article#: RR853 Number of References: 73
Title: BLOCKADE OF C5A AND C5B-9 GENERATION INHIBITS LEUKOCYTE AND PLATELET ACTIVATION DURING EXTRACORPOREAL-CIRCULATION
Author(s): RINDER CS; RINDER HM; SMITH BR; FITCH JCK; SMITH MJ; TRACEY JB; MATIS LA; SQUINTO SP; ROLLINS SA
Corporate Source: YALE UNIV,SCH MED,DEPT ANESTHESIOL,TOMPKINS 3,333 CEDAR ST/NEW HAVEN//CT/06510; YALE UNIV,SCH MED,DEPT LAB MED/NEW HAVEN//CT/06510; YALE NEW HAVEN MED CTR/NEW HAVEN//CT/06510; QUINNIPIAC COLL/HAMDEN//CT/06518; ALEX PHARMACEUT/NEW HAVEN//CT/06510
Journal: JOURNAL OF CLINICAL INVESTIGATION, 1995, V96, N3 (SEP), P 1564-1572
ISSN: 0021-9738
Language: ENGLISH Document Type: ARTICLE
Abstract: Complement activation contributes to the systemic inflammatory response induced by cardiopulmonary bypass, At the cellular level, cardiopulmonary bypass activates leukocytes and platelets; however the contribution of early (C3a) versus late (C5a, soluble C5b-9) complement components to this activation is unclear, We used a model of simulated extracorporeal circulation that activates complement (C3a, C5a, and C5b-9 formation), platelets (increased percentages of P-selectin-positive platelets and leukocyte-platelet conjugates), and neutrophils (upregulated CD11b expression), To specifically target

complement activation in this model, we added a blocking mAb directed at the human C5 complement component and assessed its effect on complement acid cellular activation, Compared with a control mAb, the anti-human C5 mAb profoundly inhibited C5a and soluble C5b-9 generation and serum complement hemolytic activity but had no effect on C3a generation, Additionally, the anti-human C5 mAb significantly inhibited neutrophil CD11b upregulation and abolished the increase in P-selectin-positive pIatelets and leukocyte-platelet conjugate formation compared to experiments performed with the control mAb, This suggests that the terminal components C5a and C5b-9, but not C3a, directly contribute to platelet and neutrophil activation during extracorporeal circulation, Furthermore, these data identify the C5 component as a site for therapeutic intervention in cardiopulmonary bypass.

8/7/28 (Item 22 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

03017755 Genuine Article#: MX293 Number of References: 27
Title: PLATELET-AGGREGATION INHIBITION BY MONONUCLEAR LEUKOCYTES
Author(s): SCHATTNER MA; FINIASZ MR; NOTRICA JA; LAZZARI MA
Corporate Source: NATL ACAD MED BUENOS AIRES,INST INVEST HEMATOL,DEPT HEMOSTASIA,PACHECO MELO 3081/RA-1425 BUENOSAIRES/DF/ARGENTINA/; NATL ACAD MED BUENOS AIRES,INST INVEST HEMATOL,DEPT TROMBOSIS & INMUNOL/BUENOS AIRES/DF/ARGENTINA/
Journal: THROMBOSIS RESEARCH, 1994, V73, N3-4 (FEB 15), P205-214
ISSN: 0049-3848
Language: ENGLISH Document Type: ARTICLE
Abstract: In this study we have investigated the effect of human mononuclear leukocytes (ML) on platelet aggregation. The results obtained demonstrated that coincubation of platelets with nonstimulated ML decreased platelet aggregation induced by collagen or thrombin in a concentration-dependent manner. The inhibitory effect increased with the incubation period of the cells, reaching a plateau at 5 minutes. T and non-T enriched ML suspensions exerted an inhibitory effect similar to the total population of ML. Supernatants from ML or mixed cell suspensions also diminished platelet aggregation. 6-keto PGF1 alpha concentration in the supernatants was less than 10 pg/ml. Hemoglobin, L-arginine and cytochrome C did not modify the antiaggregating activity of ML, whereas superoxide dismutase potentiated the inhibition of aggregation mediated by ML. The inhibitory effect was not modified by monoclonal antibody (MoAb) against the lymphocyte function-associated antigen 1, alpha subunit (LFA-1 alpha) or by a MoAb directed against ***P*** - ***selectin*** . Our results demonstrated that ML inhibited platelet aggregation, at least partially, by the release of a soluble factor(s) distinct of prostacyclin or nitric oxide. Surface adhesion molecules seem also not to be involved.

8/7/29 (Item 1 from file: 45)
DIALOG(R)File 45:EMCare

00788827 EMCare No: 30469791
The use of Cylexin (CY-1503) in prevention of reperfusion lung injury in patients undergoing pulmonary thromboendarterectomy
Kerr K.M.; Auger W.R.; Marsh J.J.; Comito R.M.; Fedullo R.L.; Smits G.J.; Kapelanski D.P.; Fedullo P.F.; Channick R.N.; Jamieson S.W.; Moser K.M.
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American Journal of Respiratory and Critical Care Medicine (AM. J. RESPIR. CRIT. CARE MED.) (United States) 2000, 162/1 (14-20)
CODEN: AJCME ISSN: 1073-449X
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 17
RECORD TYPE: Abstract

Pulmonary thromboendartectomy (PTE) for chronic thromboembolic pulmonary ***hypertension*** may be complicated by reperfusion lung injury. This has previously been demonstrated to be neutrophil-mediated. We postulated that blocking selectin-mediated adhesion of neutrophils to the endothelium with Cylexin (CY-1503) would prevent reperfusion lung injury in this patient population. In this double-blind, randomized, placebo-controlled, parallel study, 26 patients received Cylexin the day of surgery and 25 received placebo. Significantly fewer patients in the treated group (31%) compared with the placebo group (60%) developed lung injury (p = 0.036). However, the average number of days of mechanical ventilation, days in the intensive care unit (ICU) and hospital, as well as mortality were not significantly different between the treatment groups. Those with reperfusion lung injury had significantly elevated percent neutrophils, total protein, and soluble P- selectin in bronchoalveolar lavage fluid compared with those without lung injury. We conclude that reperfusion lung injury after PTE is a high-permeability lung injury and its incidence can be reduced by the administration of Cylexin on the day of surgery.

8/7/30 (Item 2 from file: 45)
DIALOG(R)File 45:EMCare

00641356 EMCare No: 29389569
Prothrombotic factors, endothelial function and left ventricular hypertrophy in isolated systolic hypertension compared with systolic-diastolic hypertension
Lip G.Y.H.; Blann A.D.; Beevers D.G.
Dr. G.Y.H. Lip, Haemost. Thrombosis Vasc. Biol. Unit, University Department of Medicine, City Hospital, Birmingham B18 7QH United Kingdom

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Journal of Hypertension (J. HYPERTENS.) (United Kingdom) 1999, 17/8 (1203-1207)
CODEN: JOHYD ISSN: 0263-6352
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 12
RECORD TYPE: Abstract

Background: Individuals with systolic-diastolic hypertension (SDH, systolic blood pressure (SBP) > 160 mmHg and diastolic blood pressure (DBP) > 90 mmHg) are at increased risk of thrombotic complications, such as stroke and heart attacks, which may be related to a hypercoagulable state. Individuals with only isolated systolic ***hypertension*** (ISH; i.e. SBP > 160 mmHg but DBP < 90 mmHg) are also at significant cardiovascular risk. We hypothesized that patients with ISH would exhibit a prothrombotic state similar to that seen in SDH. A secondary hypothesis was that individuals with ISH had similar echocardiographic parameters to those seen in SDH. Methods: We measured indices of haemorheology, endothelial dysfunction, thrombogenesis and platelet activation in 23 individuals with ISH (mean

blood pressure 193/82 mmHg), who were compared with 51 matched patients with SDH (mean blood pressure 198/112 mmHg) and 34 age- and sex- matched normotensive healthy control individuals (mean blood pressure 130/78 mmHg). Echocardiographic parameters in patients with ISH were compared to those from patients with SDH. Results: Mean plasma viscosity (an index of blood rheology, ANOVA, P= 0.001), von Willebrand factor (an index of endothelial damage, P= 0.013), plasminogen activator inhibitor-1 and lipoprotein (a) (both markers of thrombogenesis; Kruskal-Wallis test both P < 0.001) were all significantly raised in ISH and SDH relative to controls. Individuals with SDH also had high mean plasma fibrinogen (P = 0.018) and haematocrit (P = 0.010) levels compared with control individuals. There were no significant differences in levels of fibrin D-dimer or the platelet activation marker soluble P-selectin in the hypertensive patients (i.e. ISH and SDH) compared with control individuals. Patients with ISH had similar M-mode and Doppler echocardiographic parameters compared to patients with SDH. Conclusions: We conclude that individuals with ISH have abnormalities in plasma prothrombotic factors and markers of endothelial dysfunction, and echocardiographic parameters, broadly similar to that seen in SDH. This is consistent with the increased risk of thrombotic events (strokes and heart attacks) in patients with ISH.

8/7/31 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

0078220099 EMBASE No: 2000269459
Testing for endothelial dysfunction
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CORRESP. AUTHOR/AFFIL: Raitakari O.T.: Turku PET Centre, Turku University Central Hospital, PO Box 52, 20520 Turku, Finland
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Annals of Medicine (Ann. Med.) (United Kingdom) August 14, 2000, 32/5 (293-304)
CODEN: ANMDE ISSN: 0785-3890
DOCUMENT TYPE: Journal; Review RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 123

Endothelial health is a key factor in normal cardiovascular homeostasis, and recent studies have revealed several important functions of the vascular endothelium that protect against atherothrombosis. These include control over arterial tone, coagulation, fibrinolysis, and vascular growth. Consequently, endothelial dysfunction has been implicated as an important event in the pathogenesis of atherosclerosis, coronary vasoconstriction, ***hypertension*** , and myocardial ischaemia. Therefore, there has been considerable research interest in diagnostic assays for the assessment of endothelium. This review outlines the current status of markers of endothelial dysfunction, particularly those related to vasomotor control, as well as circulating markers of vascular health.

8/7/32 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

0078157261 EMBASE No: 2000206549

Vascular endothelial cell activation associated with increased plasma asymmetric dimethyl arginine in children and young adults with hypertension: A basis of atheroma?
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Blood Pressure (Blood Press.) (Norway) July 1, 2000, 9/1 (16-21)
CODEN: BLPRE ISSN: 0803-7051
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 54

The mechanism behind the development of vascular complications of ***hypertension*** in the young human remains unclear. To explore the role of vascular endothelium-generated nitric oxide (a known mediator of leucocyte- platelet-endothelial interactions) in this context, we investigated markers of endothelial activation (soluble vascular cell adhesion molecule-1 (VCAM- 1), intercellular adhesion molecule-1 (ICAM-1), P-selectin, E-selectin), and von Willebrand factor and the plasma level of the endogenous nitric oxide inhibitor asymmetric dimethyl arginine (ADMA) in a group of 31 (17 male, mean age 9.4 years) ***hypertensive*** and 9 (4 male, mean age 9.1 years) healthy, normotensive children and young adults. We found raised levels of ADMA (mean (SEM) 235 (32) n mol/l) and VCAM-1 (median (range) 1237 (675-2700) ng/ml) in the plasma of hypertensive subjects compared with those of normotensives (ADMA, 103 (7) n mol/l and VCAM-1, 1005 (425-1650) ng/ml, respectively). Furthermore, in ***hypertensive*** subjects, higher VCAM-1 concentrations ($r = 0.66$, $p < 0.001$) and vWF concentrations ($r = 0.37$, $p = 0.04$) were significantly associated with a higher plasma ADMA level. Therefore, an isolated increase in plasma VCAM-1 in hypertensives in association with raised ADMA may signify a selective 'noninflammatory' endothelial activation triggered by endothelial nitric oxide synthase inhibition. Since VCAM-1 is implicated in the origins of atherosclerosis, ADMA may be an important contributory factor in increasing the risk of atheroma formation in hypertensive children and young adults.

0078090367 EMBASE No: 2000139642
Endothelial function and hemostasis
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Zeitschrift fur Kardiologie (Z. Kardiol.) (Germany) May 1, 2000, 89/3 (160-167)
CODEN: ZKRDA ISSN: 0300-5860
DOCUMENT TYPE: Journal; Conference Paper RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 54

The vascular endothelium influences not only the three classically interacting components of hemostasis: the vessel, the blood platelets and

the clotting and fibrinolytic systems of plasma, but also the natural sequelae: inflammation and tissue repair. Two principal modes of endothelial behaviour may be differentiated, best defined as an anti- and a prothrombotic state. Under physiological conditions endothelium mediates vascular dilatation (formation of NO, PGI SUB 2, adenosine, hyperpolarising factor), prevents platelet adhesion and activation (production of adenosine, NO and PGI SUB 2, removal of ADP), blocks thrombin formation (tissue factor pathway inhibitor, activation of protein C via thrombomodulin, activation of antithrombin III) and mitigates fibrin deposition (t- and scuplasminogen activator production). Adhesion and transmigration of inflammatory leukocytes are attenuated, e.g. by NO and IL-10, and oxygen radicals are efficiently scavenged (urate, NO, glutathione, SOD). When the endothelium is physically disrupted or functionally perturbed by postischemic reperfusion, acute and chronic inflammation, atherosclerosis, diabetes and chronic arterial ***hypertension*** , then completely opposing actions pertain. This prothrombotic, proinflammatory state is characterised by vasoconstriction, platelet and leukocyte activation and adhesion (externalisation, expression and upregulation of von Willebrand factor, platelet activating factor, P-selectin, ICAM-1, IL-8, MCP-1, TNFa, etc.), promotion of thrombin formation, coagulation and fibrin deposition at the vascular wall (expression of tissue factor, PAI-1, phosphatidyl serine, etc.) and, in platelet-leukocyte coaggregates, additional inflammatory interactions via attachment of platelet CD40-ligand to endothelial, monocyte and B-cell CD40. Since thrombin formation and inflammatory stimulation set the stage for later tissue repair, complete abolition of such endothelial responses cannot be the goal of clinical interventions aimed at limiting procoagulatory, prothrombotic actions of a dysfunctional vascular endothelium.

0077988152 EMBASE No: 2000037327

Role of transforming growth factor-betal in cardiovascular inflammatory changes induced by chronic inhibition of nitric oxide synthesis

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Hypertension (Hypertension) (United States) January 1, 2000, 35/1 I (86-90)
CODEN: HPRTD ISSN: 0194-911X
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 29

We previously reported that chronic inhibition of nitric oxide (NO) synthesis with N(omega)-nitro-L-arginine methyl ester (L-NAME) induces inflammatory changes (monocyte infiltration, myofibroblast formation; and monocyte chemoattractant protein-1 [MCP-1] and transforming growth factor-beta [TGF-beta] expression) in the rat heart and vessel. There is debate regarding whether TGF-betal exhibits proinflammatory or

anti-inflammatory activities. We used the rat model to investigate the role of TGF-beta in the pathogenesis of such inflammatory changes. We show here that infiltrating monocytes and myofibroblasts in the inflammatory lesions produced TGF-beta1 on the third day of L-NAME administration. Cotreatment with a monoclonal antibody against TGF- beta1, but not with control IgG, prevented the L-NAME-induced cardiac inflammation. The antibody also significantly inhibited the gene expression of MCP-1, P-
selectin , and intercellular adhesion molecule-1. In summary, the antibody against TGF-beta1 prevented inflammatory changes in rat heart and vessel induced by chronic inhibition of NO synthesis, suggesting that increased production of TGF-beta1 is involved in the inflammatory changes in this model.

0077820111 EMBASE No: 1999306440
Neutrophil activation and mediators of inflammation in chronic venous insufficiency
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Journal of Vascular Research (J. Vasc. Res.) (Switzerland) September 9, 1999, 36/SUPPL. 1 (24-36)
CODEN: JVREE ISSN: 1018-1172
DOCUMENT TYPE: Journal; Conference Paper RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 48

The effect of venous hypertension on the state of activation of leucocytes has been investigated in patients with venous disease and control subjects. Leucocytes become 'trapped' in the circulation of the leg during periods of venous hypertension produced by sitting or standing. This is greater in the limbs of patients with chronic venous disease than controls. Studies of the plasma levels of neutrophil granule enzymes show that these are increased during periods of venous hypertension, suggesting that this causes activation of the neutrophils. Investigation of the leucocyte surface ligand CD11b shows that the more activated neutrophils and monocytes are sequestered during venous
hypertension . Measurement of plasma levels of the soluble parts of the vascular (VCAM), intercellular (ICAM) and endothelial leucocyte (ELAM) adhesion molecules show that these are all elevated in patients with chronic venous disease compared to controls. Following 30 min of venous hypertension produced by standing, these levels are further increased. These data suggest that venous ***hypertension*** causes neutrophil and monocyte activation, which in turn causes injury to the endothelium. I believe that this may be the mechanism that initiates the pathological processes which lead to venous ulceration. It has recently been shown that the venotonic drug Daflon 500 mg (450 mg diosmin, 50 mg hesperidin, Servier, France) influences these processes. Surface expression of CD62L is reduced in neutrophils and monocytes, and plasma levels of soluble endothelial adhesion molecules are reduced. These observations may explain the antiinflammatory effects of Daflon 500 mg.

8/7/36 (Item 6 from file: 73)
DIALOG(R)File 73:EMBASE

0077377941 EMBASE No: 1998288237
Increased fraction of circulating activated platelets in acute and previous cerebrovascular ischemia
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Thrombosis and Haemostasis (Thromb. Haemost.) (Germany) August 1, 1998 , 80/2 (298-301)
CODEN: THHAD ISSN: 0340-6245
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 29

Determination of circulating activated platelets may be helpful to estimate the prognosis and to stratify therapies in arterial vascular disorders including stroke. We used flow cytometry and phase contrast microscopy to study whether the fraction of platelets expressing p-selectin and CD63 and the fraction of platelets with shape change are increased in patients with acute and previous cerebrovascular ischemia. The proportion of platelets expressing activation dependent antigens was higher in patients with acute (n = 24; p-selectin: 8.23 +/- 4.21%; CD63: 3.53 +/- 2.53%) and with previous cerebrovascular ischemia (n = 46, 3.86 +/- 1.98%; 2.80 +/- 1.79%) as compared to age- and sex-matched control subjects (n = 35; 2.17 +/- 0.95%; 1.79 +/- 0.75%; p <= 0.005, respectively). In patients with previous ischemia, there was no difference between treatment with aspirin (n = 25) or phenprocoumon (n = 21). ***Hypertension*** , diabetes mellitus and smoking were not associated with increased antigen expression (analysis of variance). The fraction of discoid platelets and platelet counts were not significantly different between groups. Our results indicate increased expression of platelet neoantigens in acute and to a less degree in previous cerebrovascular ischemia. Ongoing platelet activation after cerebrovascular ischemia despite therapy with aspirin or phenprocoumon indicates that new anti-platelet drugs may be of benefit for these patients. Flow cytometry appears to be a useful tool to assess platelet function in cerebrovascular ischemia.

8/7/37 (Item 7 from file: 73)
DIALOG(R)File 73:EMBASE

0077051577 EMBASE No: 1997344846
Evidence of platelet activation in hypertension
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Journal of Human Hypertension (J. HUM. HYPERTENS.) (United Kingdom)

November 20, 1997, 11/9 (607-609)
CODEN: JHHYE ISSN: 0950-9240
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 5

To test the hypothesis that platelet activation is present in hypertension, we measured plasma markers beta thromboglobulin and soluble P-selectin in hypertensive patients and normotensive controls. Both markers were raised in the patients (P < 0.05), and in a subgroup of patients, beta thromboglobulin was reduced with successful treatment of ***hypertension*** with the ACE inhibitor quinapril. We suggest that reversible platelet activation is present in ***hypertension*** . This may be a contributing factor to the link between this risk factor and the development of thrombotic disease such as stroke.

8/7/38 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13920592 PMID: 11789207
[Effect of yimai jiangya extract on platelet activation and fibrinolytic activity and angiotensin II in aged patients with essential hypertension]
Duan X; Yang D; Sun X
Jinan Military Regional General Hospital, Jinan (250031).
Zhongguo Zhong xi yi jie he za zhi Zhongguo Zhongxiyi jiehe zazhi = Chinese journal of integrated traditional and Western medicine / Zhongguo Zhong xi yi jie he xue hui, Zhongguo Zhong yi yan jiu yuan zhu ban (China) Jul 2000, 20 (7) p508-10, ISSN 1003-5370--Print
Journal Code: 9211576
Publishing Model Print
Document type: Clinical Trial; English Abstract; Randomized Controlled Trial
Languages: CHINESE
Main Citation Owner: NLM
Record type: MEDLINE; Completed
OBJECTIVE: To observe the effect of Yimai Jiangya extract (YMJYE) on platelet alpha-granule membrane protein (GMP-140), tissue-type plasminogen activator (t-PA) and its inhibitor (PAI), angiotensin II (AngII) in aged patients with essential hypertension (EH) of stage 2 caused by Qi deficiency and blood stasis. METHODS: Radioimmunoassay (RIA) and colorimetric analysis were used to examine the levels of GMP-140, t-PA, PAI and AngII before and after treatment with YMJYE in 42 aged patients (treated group), compared with that of 30 aged patients before and after treatment with captopril (control group) and 30 aged healthy subjects (healthy group). RESULTS: Before ***treatment*** , ***GMP*** - ***140*** , PAI and AngII were significantly higher (P < 0.01), and t-PA were obviously lower (P < 0.01) in the treated and the control group compared with those in the healthy group. After ***treatment*** , the improvement of GMP-140, t-PA, PAI and AngII in the ***treated*** group were obvious (P < 0.05, P < 0.01). The improvement of GMP-140, t-PA, PAI in the treated group was more significant than that of control group (P < 0.05, P < 0.01), while the improvement of AngII in the treated group was worse than that of the control group (P < 0.01). After treatment, there was insignificant changes about the levels of blood pressure between the treated group and the control group (P > 0.05). CONCLUSIONS: In the aged patients, GMP-140, PAI and AngII were increased, t-PA reduced. The effects of YMJYE in inhibiting platelet activation and improving fibrinolytic activity were better than

that of captopril. These findings indicated that the mechanisms of inhibiting platelet activation and improving fibrinolytic activity and lowering blood pressure of YMJYE were correlated with decreasing the levels of AngII, but other mechanisms may also exist.
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8/7/39 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13769292 PMID: 11031209
High plasma serotonin levels in primary pulmonary ***hypertension*** . Effect of long-term epoprostenol (prostacyclin) therapy.
Kereveur A; Callebert J; Humbert M; Herve P; Simonneau G; Launay J M; Drouet L
CR C. Bernard "Pathologie Experimentale et Communications Cellulaires", IVS and IFR 6, Biochimie et Angio-Hematologie, Hopital Lariboisiere, AP-HP, Paris, France.
Arteriosclerosis, thrombosis, and vascular biology (UNITED STATES) Oct 2000, 20 (10) p2233-9, ISSN 1524-4636--Electronic
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Elevated plasma serotonin is associated with primary pulmonary ***hypertension*** (PPH). To test whether this elevation could be related to platelet activation, the 2 pools of blood serotonin (platelets and plasma) and plasma 5-hydroxyindoleacetic acid (5-HIAA) as well as markers of platelet activation (alpha(IIb)beta(3), CD36, P-selectin, and CD63 membrane epitopes) were measured in 16 patients with severe PPH (group 1) before and at days 10 and 40 of treatment with a continuous infusion of epoprostenol (prostacyclin). The same biological parameters were also measured in 19 healthy subjects (group 2) and in 10 patients after cardiovascular surgery with extracorporeal circulation (group 3), a condition known to profoundly activate the platelets. Twelve PPH patients showed hemodynamic and clinical improvement, 3 remained stable, and 1 had the treatment stopped because of clinical aggravation. At day 0, mean plasma serotonin (5-hydroxytryptamine [5-HT]) concentration was much higher in PPH patients than in normal subjects (34.4+/-21.2 versus 9.1+/-6.0 nmol/L, respectively; P:<0.001) and positively correlated with total pulmonary resistance. The mean platelet 5-HT content was not significantly different in PPH compared with normal individuals. Mean plasma 5-HIAA concentrations were much higher in PPH than in normal patients (162+/-57 versus 61+/-7 nmol/L, respectively; P<0.001). These parameters did not significantly change during epoprostenol treatment. There was no correlation between the changes in plasma 5-HT during treatment and clinical or hemodynamic improvement. In PPH patients, the mean platelet volume significantly decreased (ANOVA, P<0.01) during treatment. Positive correlations were evidenced between the size of platelets and the number of alpha(IIb)beta(3) and CD36 epitopes. When compared with control platelets, the number of alpha(IIb)beta(3) epitopes detected on PPH platelets at day 0 tended to be higher, but this difference did not reach a statistical significance (41 300+/-7140 for PPH patients versus 36 010+/-3930 for control subjects, P=0.069). The number of CD36 epitopes, in the range of controls at day 0 (11 590+/-5080 for PPH patients versus 11 900+/-1790 for control subjects), decreased during treatment (ANOVA, P=0.038) and became significantly low at day 40 (8660+/-3520, P<0.001). The number of CD63

epitopes was not elevated, and P-selectin was never detected at any time point on PPH platelets. This glycoprotein profile indicates that the platelets of PPH patients were not highly activated but had an accelerated turnover and returned to normal under epoprostenol treatment without change of the elevated plasma serotonin, characteristic of PPH. In conclusion, neither platelet activation nor a significant alteration of the 5-HT endothelial metabolism explains the high level of plasma 5-HT in PPH patients. The 5-HT plasma concentration is not a predictive marker of the severity of PPH, and its evolution is independent of the clinical and hemodynamic status. Treatment by a potent antiaggregating agent, epoprostenol, does not affect the increase of plasma 5-HT, despite a therapeutic benefit.
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DIALOG(R)File 155:MEDLINE(R)

11216364 PMID: 7533336
Amyloid beta-protein precursor-rich platelet microparticles in thrombotic disease.
Nomura S; Komiyama Y; Miyake T; Miyazaki Y; Kido H; Suzuki M; Kagawa H; Yanabu M; Takahashi H; Fukuhara S
First Department of Internal Medicine, Kansai Medical University, Osaka, Japan.
Thrombosis and haemostasis (GERMANY) Oct 1994, 72 (4) p519-22, ISSN 0340-6245--Print Journal Code: 7608063
Publishing Model Print
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We investigated the association of amyloid beta-protein precursor (APP) and platelet derived microparticles in 20 normal controls and 91 patients with various diseases causing a thrombotic tendency. Compared with the controls, the mean percentage of APP-positive microparticles was significantly greater in the patients with cerebral infarction (39.1 +/- 17.7%, p < 0.001), diabetes (31.1 +/- 12.6%, p < 0.001), and uremia (30.1 +/- 14.7%, p < 0.01), but not in those with ***hypertension*** (8.2 +/- 6.3%, p = NS). Sixteen patients with cerebral infarction, 20 with diabetes, and 11 with uremia had microparticles with very high APP levels. In normal controls, 7.2 +/- 3.7% of the microparticles were positive for P-selectin, while the percentage in cerebral infarction, diabetes, uremia, and ***hypertension*** was respectively 43.5 +/- 15.1%, 40.0 +/- 12.8%, 31.8 +/- 12.2%, and 11.6 +/- 7.3%. There was a significant correlation between P-selectin and APP positivity of microparticles. Our results suggest that microparticle APP may have a regulatory influence on coagulation abnormalities.
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8/7/41 (Item 1 from file: 370)
DIALOG(R)File 370:Science

00500312 (USE 9 FOR FULLTEXT)
Molecular Therapies for Vascular Diseases

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Abstract: Vascular disease is the most common cause of death in the industrialized world. Although significant progress has been made in treating these disorders, more therapeutic agents must be developed that effectively prevent, arrest, or reverse this disease. Recent insights into the pathogenesis of vascular disease have opened up a new frontier of molecular therapies that target molecules as diverse as adhesion molecules and transcription factors. The biological rationale for these new therapies and their prospects for success are discussed.
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Dehydroepiandrosterone derivatives for preventing progressive tissue necrosis reperfusion injury, bacterial translocation and adult respiratory distress syndrome
INVENTOR(AUTHOR): Araneo, Barbara A.; Orlinska, Urszula; Farrukh, Imad S.; Daynes, Raymond A.
LOCATION: USA
ASSIGNEE: Paradigm Biosciences, Inc.; University of Utah Research Foundation
PATENT: PCT International ; WO 9640152 A1 DATE: 19961219
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SECTION:
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IDENTIFIERS: dehydroepiandrosterone ischemia damage prevention, reperfusion injury prevention dehydroepiandrosterone, bacterial translocation prevention dehydroepiandrosterone, adult respiratory distress syndrome dehydroepiandrosterone
DESCRIPTORS:
Chemistry...
chems., burns from; dehydroepiandrosterone derivs. for preventing or reducing the effects of ischemia assocd. with trauma, bacterial translocation, ARDS, blood cell adhesion, and pulmonary hypertensio
Platelet aggregation inhibitors... Platelet(blood)... P-selectin...
Vascular endothelium...
dehydroepiandrosterone derivs. for inhibiting P-selectin expression by platelets and endothelial cells
Adult respiratory distress syndrome... Antihypertensives... Anti-ischemic agents... Blood cells... Burn... Cell adhesion... Hemorrhagic shock...
Intestinal bacteria... Myocardial infarction... Necrosis... Neutrophil adhesion... Platelet adhesion... Pulmonary hypertension... Reperfusion injury... Sepsis...
dehydroepiandrosterone derivs. for preventing or reducing the effects of ischemia assocd. with trauma, bacterial translocation, ARDS, blood cell adhesion, and pulmonary hypertension
Antiatherosclerotics...
dehydroepiandrosterone derivs. for preventing or reducing the effects of ischemia assocd. with trauma, bacterial translocation, ARDS, blood cell adhesion, and pulmonary hypertension and other diseases
Vascular injury...
from reperfusion; dehydroepiandrosterone derivs. for preventing or reducing the effects of ischemia assocd. with trauma, bacterial translocation, ARDS, blood cell adhesion, and pulmonary hypertension
Heart diseases...
hyperactive circulation; dehydroepiandrosterone derivs. for preventing or reducing the effects of ischemia assocd. with trauma, bacterial translocation, ARDS, blood cell adhesion, and pulmonary hypert
Trauma...
surgical and accidental; dehydroepiandrosterone derivs. for preventing or reducing the effects of ischemia assocd. with trauma, bacterial translocation, ARDS, blood cell adhesion, and pulmonary hypert
Surgery...
trauma; dehydroepiandrosterone derivs. for preventing or reducing the effects of ischemia assocd. with trauma, bacterial translocation, ARDS, blood cell adhesion, and pulmonary hypertension
CAS REGISTRY NUMBERS:
534-30-3D congeners, dehydroepiandrosterone derivs. for preventing or reducing the effects of ischemia assocd. with trauma, bacterial translocation, ARDS, blood cell adhesion, and pulmonary hypertension
534-30-3 5211-75-3 6514-89-3 10939-10-3 12327-31-3 1051199-68-3
dehydroepiandrosterone derivs. for preventing or reducing the effects of ischemia assocd. with trauma, bacterial translocation, ARDS, blood cell adhesion, and pulmonary hypertension
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